THE
# LARYNGOSCOPE.

VOL. XXXIX FEBRUARY, 1929. No. 2

## ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

### LARYNGO-RHINOLOGY AND GENERAL MEDICINE.*†

PROF. DR. M. HAJEK, Vienna.

Your invitation to lecture today fills me with pride and satisfaction for two reasons: First, I am glad that, because of this honored request, it is possible for me to renew more intimately the old friendly relations with my English professional colleagues, which date back more than 35 years; secondly, it affords me very special satisfaction to be permitted to dedicate a few words of remembrance to the memory of Sir Felix Semon—to the man who rendered immortal service in the realm of laryngology, and for whom, moreover, I have personally preserved a particular feeling of gratitude, inasmuch as he helped me in my own home to the best of his ability.

In keeping with the mental outlook of Sir Felix Semon, which extended far beyond what he expressed through laryngo-rhinology alone, I have chosen a general theme which is qualified to produce thought about the development of our specialty, and perhaps also profitable stimulation. You must permit me, however, to review the development of our specialty up to the present, before entering upon the actual subject of my lecture.

The diagnosis of diseases of the upper air passages made possible through the epoch-making accomplishments of Garcia, Türck and Czermak founded the specialty of laryngo-rhinology. It is easy to understand that this new field of medicine attracted many fellow-

*The Semon Lecture, 1928, of the University of London, delivered on Nov. 1 at the Royal Society of Medicine, London.

†Republished by special courtesy of the Journal of Laryngology and Otology, January, 1929.

workers ready to reap with relatively little trouble the harvest of laurels from a previously untilled soil. The rapid advances in diagnosis and in the instrumental technique going parallel with it, intoxicated the first generation; thus it came about that our specialty ran the risk of losing its links with general medicine.

I should like to mention only one significant example as an illustration of the pernicious results which supervened upon these conditions. The older amongst you will remember, as I do, the time when curettage of tuberculosis of the larynx was introduced by Heryng and Krause and was adopted by many laryngologists, who practiced it without any regard for the general condition of the patient. All of us, with few exceptions, took some part in this line of treatment. I well remember demonstrating in my clinic the healed scars on the interarytenoid mucous membrane of a female patient, age 24 years, whom I had curetted. The enthusiasm over the case gave way after a few days to a general feeling of disenchantment, when we were informed that she had died of systemic tuberculosis. At that time I coined the expression: "The larynx healed, the patient killed".

This and similar failures soon led to a change in outlook. We know today that no diseased condition in the upper air passages can be correctly estimated without consideration of the whole system. It is superfluous before such an audience as this to allude to the progress which followed the co-operation of the laryngologist with the physician, surgeon, ophthalmologist, neurologist and others. It is unnecessary to quote examples in this connection.

What then is the relation today of the special branches—especially laryngo-rhinology—to general medicine?

Recognizing the inseparable correlation between the local manifestations and general disease, we try to complete and confirm the nature of the laryngoscopic and rhinoscopic picture with the aid of those specializing in the adjoining areas, and with the help of the laboratory, by means of histological and serological examinations and by the application of the X-rays. Who would deny nowadays that the throat clinic without the above-mentioned auxiliary sciences was in many instances like a lonely island, itself barren, and maintaining life only through importation from without! The youngest amongst us cannot envisage a clinic without these auxiliary sciences; and yet that was the condition of things only a few decades ago.

Having pointed out the relations prevailing at the present time between our specialty and general medicine, it is now my duty to consider once more whether we have reached the end of our resources,

and if the relationship deserves to be definitely preserved. In the first place, however, one further observation.

Every clinician knows that the signs of disease present an ever-changing polymorphism and only the most typical appearance makes the diagnosis absolutely certain. In between, there are many changes from the normal form which do not permit us to make a definite statement. Let me give two examples: We recognize with certainty a normal larynx and just as certainly an advanced condition of tuberculosis of the larynx; but in between there are a great number of indefinite forms that we are not able to interpret *a priori* with absolute certainty. The same is the case with regard to our opinion as to a normal vocal cord and a case of pronounced cancer of the same, but between the extreme forms there are many abnormal appearances, none of which are absolutely characteristic.

With the aid of the laboratory methods now in use we can diagnose these somewhat indefinite clinical pictures of disease; since these methods are doubtless able to do this often, as a rule, they will be valued for the most part as the deciding factor in diagnosis. We as clinicians, therefore, have gradually accustomed ourselves to underestimate the value of our clinical experience and to expect salvation entirely from the auxiliary sciences.

In this lies, in my opinion, the crisis in all medicine at the present day; it urgently needs to be combated if we do not wish to run the risk of being deprived of the blessings derived from the century-old practice of clinical medicine.

The overuse of laboratory diagnosis for clinical purposes is the result of the, as yet, little-opposed view that, while clinical manifestations may present uncertainties in diagnosis, more or less absolute power of proof belongs to the laboratory methods. But this is by no means the case. The same holds true also in regard to the latter; they may supply diagnostic information of absolute, of relative, and often enough, of no value at all.

The information which the laboratory provides is only one aspect of the disease-complex. Its significance depends upon how it is estimated in the particular case, and this is for the clinician to decide.

In order to make this viewpoint understandable in its entire scope, I should like to give a few significant examples from my experience, in connection mainly with the use of histology, roentgenology and serology.

*Histological Examination:* Who will deny that histological examination today comprises one of the most important and indispensable means for clinical purposes? In very many cases, it alone gives absolute proof of the character of the diseased process. This has gradually led us to regard histological diagnosis as an always "infallible" means. It is meanwhile forgotten that the histological diagnosis is the product of a more or less logical series of single histological pictures, whose value is variously estimated even by the histologists. Accordingly, in the great variety of pictures, the diagnosis reflects more the view of the histologist than the infallible truth. From this are derived the many false histological values which may confuse the clinical picture, as many of us still believe in the infallibility of the histological report.

A few examples may be given: I have noted in my records three cases of carcinoma of the larynx, with periods of observation covering 25, 16 and 10 years. In all three cases, there was thickening of one vocal cord in adults. The histological diagnosis from the biopsy in all of them was that of epithelial carcinoma, with the special additional note in one case, that the epithelial cells were already in the lymph vessels. My proposal to split the larynx and perform radical extirpation was refused by the patients, but they have remained well until the present day; in two of them the thickening has entirely disappeared; in one it exists without further change. Furthermore, in a case of removal of the larynx, in spite of the positive histological diagnosis of carcinoma, the condition later proved to be tuberculous.

Since the occurrence of these false diagnoses, I accustomed myself to obtain a clearer insight into the history of carcinoma, and found that it is true, that the marked histological variations in carcinoma are at once proof that there are numerous pictures which are certainly suspicious, but nevertheless are not those of carcinoma. The histologists know these pictures quite well, but are not agreed among themselves as to what shall be designated as only "suspicious", and what as "true carcinoma". My deceased friend and patron, Prof. Paltauf, who, in addition to his thorough professional knowledge, was distinguished by a commensurate conscientiousness, admitted to me that he was governed by the greatest caution in making a diagnosis from tissue examination. Although 25 years ago he believed that he could almost always make a correct diagnosis, during the last years of his activity he became wiser from experience, and refrained from the diagnosis of "carcinoma". He especially emphasized that fragments from areas previously cauterized or irritated in another way, very often displayed typical epithelial cones with distinct cell meta-

morphosis, which could be very similar in appearance to cancer without actually being so. But the best proof that this kind of microscopic picture can be variously interpreted is the circumstance which has occurred in my own experience, that one and the same histological picture is interpreted differently by different histologists. This experience alone must teach us as clinicians that we must take a histological appearance that is not absolutely unequivocal *cum grano salis,* especially if the clinical picture is doubtful as to such a postulation. In these cases it is better to wait and later make repeated excisions, until the picture is absolutely positive or negative.

Allow me to dwell upon some preparations to illustrate what has been said.

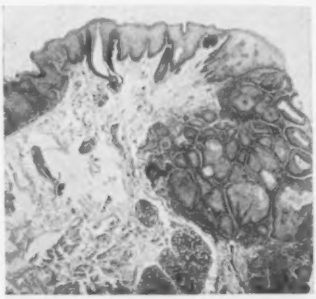


Fig. 1. Pachydermia of buccal mucous membrane; on the right side the histological picture is that of carcinoma.

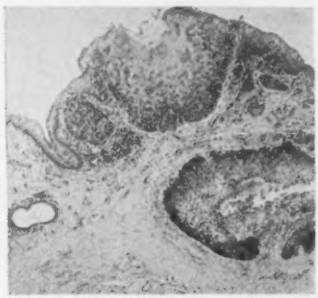


Fig. 2. Carcinoma of the vocal cord.

Fig. 1 is a preparation of a high-power view of pachydermia of the buccal mucous membrane, which appeared broken up at one place, and was therefore suspicious of commencing carcinoma. On one side of this preparation there is seen the marked thickening of the epithelium with cones, which penetrate deeply into the connective tissue layer. But in spite of this penetration, the cones are clearly outlined and the epithelium regularly arranged. One can say with certainty that these epithelial plugs, penetrating deeply, show no evidence of a commencing carcinoma; one can, moreover, say regarding the cornification of the surface epithelium that it represents typical pachydermia. One can state with equal certainty that the irregularly penetrating epithelial plugs on the right side of the preparation, whose edges are bordered by anaplastic epithelium, give a certain picture of

carcinoma. Fig. 2 is a preparation of carcinoma of the vocal cord in which the limit between the normal tissue and the cancerous part is as sharp as in the former preparation. On the right there is quite normal epithelium, on the left equally characteristic carcinomatous tissue. Between these two extremes—characteristics in a positive as well as in a negative sense—there are a great number of pictures which, to be sure, awaken suspicion of commencing carcinoma, the proof of which, however, cannot be established.

Fig. 3 is suspicious of carcinoma because of the deep penetration of the epithelial plugs. The cells show anaplasia and the plugs are irregularly defined against the connective tissue. However, the fact that we do not find anywhere in the deep parts detached portions of

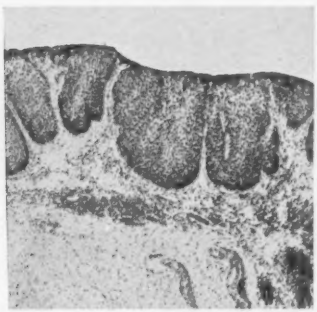


Fig. 3. Deep penetration of epithelial cones, the cells showing anaplasia and the cones irregularly defined against the connective tissue.

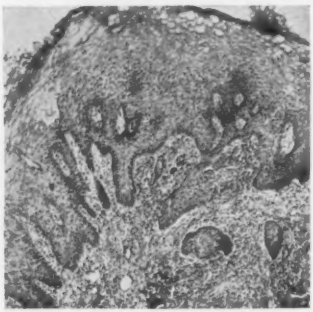


Fig. 4. A case of tuberculosis of the larynx in which the surface epithelium penetrates deeply, simulating the change in carcinoma.

the plugs does not allow us to make a positive diagnosis of cancer. It is the kind of preparation on which different histologists give different opinions, inasmuch as many assert the great probability of carcinoma, while others consider the evidence is negative. In this case, the correct diagnosis of carcinoma was made from the microscopic examination of another specimen of tissue on the basis of more characteristic changes.

Even deeply burrowing and detached epithelial cones, which are very similar to what is found in carcinoma, can deceive. This is seen in Fig. 4 from a typical case of tuberculosis of the larynx. Here in different places, the surface epithelium is greatly proliferated, penetrating deeply into the connective tissue and forming atypical epi-

thelial cones, in a manner which we are accustomed to see only in typical carcinoma. In tuberculosis, however, the epithelium seems to assume an often strongly hyperplastic form, and to construct a branched net of epithelial cords in the deeper tissues. Accordingly, we find in the literature a large number of cases, in which the microscopical examination *in vivo* showed typical carcinoma, while after the extirpation of the larynx, an undoubted tuberculosis could be established.

A further example which occurred in my clinic recently is especially instructive. A man had multiple coalescing tumors of the tongue, with moderately infiltrated surrounding tissue and soft granulations in the neighborhood of the ulcerations. The most probable clinical diagnosis was that of tuberculosis. The patient was already under treatment in other hands, and brought with him the report of a strongly positive Wassermann reaction, although the ulcers did not have the least resemblance to gummatous ulcers. The histological examination of several pieces excised from different places showed diffuse granulation tissue with numerous epithelioid cells and Langerhans' giant-cells. The histologist was of the opinion that this spoke with certainty neither for tuberculosis nor lues, but the fact that the giant-cells in numerous stained sections never contained tubercle bacilli, together with the serological result, established the diagnosis quite certainly in favor of lues. I determined upon antiluetic treatment reluctantly, inasmuch as I, as a relic of the old school, still regarded the ulcers of the tongue as tuberculous. During the ensuing, unavailing antiluetic treatment, driven by doubt, I removed several more pieces from the smooth edges of the ulcers; in these I succeeded in finding beyond all doubt in several sections giant-cells and tubercle bacilli. The subsequently instituted local light treatment with the Wessely lamp had brought about in a short time a definite improvement. In this case, the clinical observation, that is the macroscopical appearance of the ulcers, corrected the result of the serological and histological examinations.

As further commentary, however, there is still the following to be added: The positive Wassermann proved that the patient was originally luetic, but the lues had nothing to do with the formation of the ulcers on the tongue. The histologist had no reason to diagnose syphilis on the basis of the histological examination since there was no absolute proof either of syphilis or of tuberculosis. The histologist philosophized, and thereby overstepped his bounds.

These and numerous similar occurrences make it clear to the clinician that he must control the histological diagnosis. But in order to

be able to do this successfully, he himself must have experience of the fundamental points of histological diagnosis. Particularly must he be clear as to whether the histology in question can be considered conclusive of the stated diagnosis. If he is not able to do this, then he runs the risk of falling into false pathways.

*Serological Examination:* In this connection, I have already given an example with the last case. It should surely never be forgotten that the serological examination furnishes only a single, and not even a safe, link in the chain of evidence which makes certain the clinical diagnosis. We laryngologists especially must hold fast to our principle that the clinical diagnosis, founded by our predecessors with great diligence and acuity, affords an incomparably stronger support to us, than that based on serological examination. The Wassermann reaction and all other reactions connected with syphilis prove after all, in any given case, only, whether syphilis is present in the body. But whether, in spite of a positive reaction, the pathological change in question is tuberculous, luetic or carcinomatous, for that, it proves nothing; without mentioning the fact that a negative examination embodies no proof whatsoever.

*Radiological Examination:* The sources of error dependent upon radiological pictures are legion. Radiology, however, is not to blame, but rather the estimation in which it is held as a means of diagnosis. It is a naturally human ambition to make every auxiliary method of diagnosis indispensable and superior and to place it in the brightest light. In this effort the goal is often overshot. It is again the business of the clinician to place the proper value upon the skiagrams provided. In doing so, it is more practical to pay attention to the actual picture than to the finished diagnosis. If this is not done, one can sometimes have an unfortunate experience, as happened to me in the following case, eight years ago, at a time when radiological diagnosis was still a dogma for me.

A boy, age 14 years, was brought to my clinic from a department of the hospital, with the statement that there was a question as to the presence of a foreign body in the esophagus. Several unsuccessful attempts at removal had been made, as the many wounds and considerable swelling of the esophagus testified. An accompanying X-ray plate showed ostensibly the foreign body wedged in the esophageal wall at the level of the bifurcation of the trachea. The esophagoscopic examinations, repeatedly made in my clinic both by myself and by my assistants, did not detect a foreign body either in the lumen or in the, to be sure, greatly mutilated and swollen esophageal wall.

But the supposition persisted that manifestly the foreign body was present in the esophageal wall . The lungs were entirely normal. Another X-ray picture confirmed the above-mentioned position of the metallic foreign body, and the radiologist insisted thereupon that the foreign body must be in the esophageal wall. However, further attempts at removal were also negative. Within a few days the patient had fever, right-sided purulent exudate in the pleura, with bilateral bronchopneumonia. The necropsy showed a foreign body in the entrance to the right bronchus, as may be soon in Fig. 5.

I should like to comment upon this case in the following manner. There was no esophageal foreign body at all, but on the contrary a

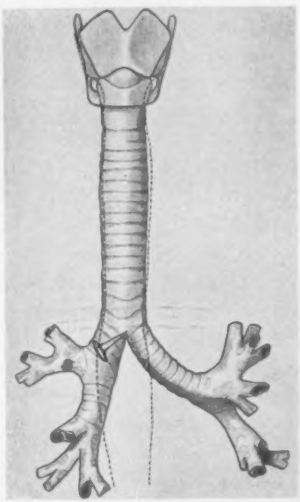


Fig. 5. Foreign body at entrance to the right bronchus.

Fig. 6. Tumor in lower part of the trachea.

Fig. 7. On the left, fragments of the tumor, and on the right the main mass.

bronchial foreign body. The wounds in the esophagus were altogether of a traumatic nature, as a result of the previous esophagoscopic interference. The Roentgen picture was correct but falsely interpreted, since a foreign body lying at the level of the bifurcation can belong equally to the esophagus and to the bronchial tube, as we already knew and taught.

In this case, therefore, the diagnosis of a foreign body in the esophagus, established definitely and decidedly by two Roentgenolo-

gists, was not only false, but it also led us into error by confusing us. Without the misleading diagnosis of the radiologists, the foreign body would not have escaped us. A book could be written about the numerous deceptions of radiology in the realm of laryngo-rhinology. Everyone of us must have had our own experience in this connection. I should like to cite the statement of a prominent bronchoscopist, Mann, who in his compilation of cases, makes the crushing avowal that up to now Roentgenology has brought more misfortune than success in the pathology of the air passages. If this is the case, then it is not due to the roentgenological pictures as such, for they are always correct, but rather to their incorrect interpretation or evaluation, with a faulty consideration of the entire clinical symptom-picture.

I do not wish to be misunderstood and awaken the belief that I question the value of the laboratory diagnoses and at times their issue-deciding significance; I only wish them to have their correct place in the series of diagnostic measures that come under consideration. But when I see everywhere in the practice of today, how these methods are overestimated in the clinics and how often, instead of expediting, rather obscure clinical diagnosis, then I believe I must express the fear that clinical medicine will be pushed unjustly into the background by laboratory diagnoses.

Figuratively, I should like to compare a clinical diagnosis with a symphony orchestra, in which every single instrument has its proportionate significance. No instrument is allowed to come into prominence unduly, unless it is called upon to produce the leading melody. The director has to restrain every existing excess, so that the musical effect of the whole may suffer no loss. I hope that the analogy is understandable.

It seems to me, therefore, that it is the clinician's task, if his efforts are to be accompanied by results, to criticize both the value and the reliability of the laboratory examination. But in order to be able to do this, he must be well versed in the above-mentioned spheres, and be able sometimes to make the examinations himself. He must at least be sufficiently acquainted with them to be able to differentiate the certain from the doubtful. There is no standing still in the knowledge of a clinician; there is only a "forwards" and a "backwards"; he must continually learn if his efficiency is to remain on the height of progress.

What has been said of the relations of laryngo-rhinology to the laboratory sciences applies equally to most of the other special

branches of medicine. In this there will probably be no essential difference.

Laryngo-rhinology, meanwhile, has still further very important connections with the great branches of medicine; internal medicine and surgery require a few words.

Since the pathological changes in the trachea and bronchi have become known through endoscopy, the recognition of their relation to the organs within the thorax has become especially urgent. I mention only the changes in the deeper layers of the trachea through diseases of the mediastinum, bronchial carcinoma, bronchiectases, and lung abscesses resulting from foreign bodies and the manifold

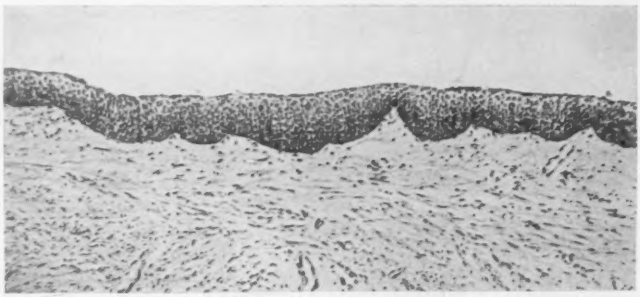


Fig. 8. Microscopic section of the tumor in Figs. 6 and 7, showing its fibrous character.

inflammatory processes of the bronchial tree, in conjunction with the pathology and therapy of the air passages.

The knowledge of the pathology of the thoracic organs was recognized by the pioneers of our specialty as a neighboring province. Today, however, a superficial knowledge is no longer enough. It is not sufficient that the internist and laryngologist should work side by side, that is, that they reach their conclusions separately; they must attack together the problem at hand—and every case of sickness is a special problem—they must stimulate each other, advise and correct. I shall quote two examples from my clinical experience which will illustrate this.

A man, age 74 years, was sent to me by his family physician, on account of marked dyspnea, and at the same time the following facts, already obtained by the internist and a laryngologist, were communi-

cated. In both lungs there was present moderate diffuse bronchitis without decreased resonance, without rise of temperature, and with moderate emphysema. In the larynx and trachea, slight catarrh without deeper changes was present. Since the dyspnea was proportionately of high degree, the family physician felt that the catarrhal condition did not satisfy him, that there must still be something further in the middle or deep air passages causing constriction. My examination showed, in addition to the described moderate changes in the lungs, a perfectly passable larynx and trachea; the most inferior part

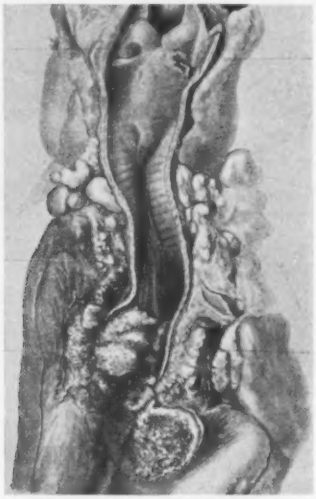


Fig. 9. Primary carcinoma of right bronchus; metastases in larynx and right lung; carcinomatous bronchial glands.

of the latter could not, however, be seen by indirect examination. It seemed to me, nevertheless, as if there were something uneven present in the deepest part of the trachea, without, however, being able definitely to prove it. The family physician urged a more comprehensive examination, which was possible only by means of direct tracheoscopy. Fig. 6 shows by this examination a tumor nearly as large as a walnut in the deepest part of the trachea, lying immediately over the bifurcation and attached to the right tracheal wall; the entrance to both bronchi proved to be markedly narrowed, so that only a small slit was left, as seen in the illustration. In spite of broad adhesions,

I removed the tumor by the direct method. Three small pieces (Fig. 7) were first removed, in order to widen the respiratory passage, and only then was I able to remove without danger the larger piece. The tumor proved to be a pure fibroma, as seen in Fig. 8. There is on the surface stratified squamous epithelium with a distinct basement membrane, beneath which there is fibrous connective tissue with blood vessels and numerous fibroblasts in different stages of development. Two years have already elapsed since the operation and the patient remains perfectly well.

The merit in the clearing up of the case is due to the general physician alone, who, through his comprehensive view of the entire disease-process, forced its recognition. The examinations of the laryn-

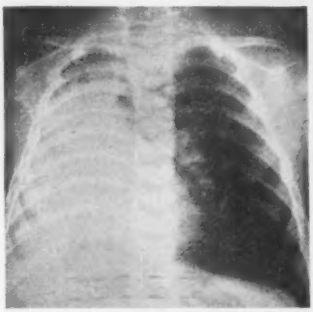


Fig. 10. Atelectasis of right lung due to foreign body in right bronchus; trachea and heart displaced to the right.

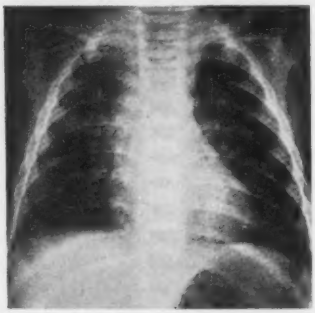


Fig. 11. After removal of foreign body, right lung is almost completely clear, with trachea and heart in normal position.

gologist and internist working *side by side* and not *together* were not able to accomplish this.

A second case is no less instructive. A patient, age 50 years, was brought into my clinic on account of hoarseness and dyspnea. The examination showed in the subglottic area a flat, uneven tumor of the right side of the larynx, by which, however, respiration could not have been seriously disturbed. As the more probable cause of the dyspnea, there could be established complete dullness of the left chest, with displacement of the heart and vicarious emphysema of the right lung. Roentgenologically, complete infiltration of the left lung was confirmed. There had never been fever. The cause of the lung disease could not be determined. The subsequent tissue examination of the subglottic tumor showed undoubted carcinoma. The relation of

the small carcinoma of the larynx to the completely infiltrated left lung could not be cleared up at once. To be sure, it was almost impossible that it was a question of metastasis, inasmuch as: *1.* according to experience, metastases do not occur in early intralaryngeal carcinoma, and, *2.* there was no swelling of regional lymph glands present in the neck, which would undoubtedly have been present if it had been a question of a later metastasis in the lung. It was now evident that the information at hand did not suffice for a satisfactory solution of the case. I determined, therefore, in spite of the dyspnea present to carry out a careful direct bronchoscopy, which showed a large tumor in the left bronchus. The tumor made the left bronchus totally impassable; it was uneven and ulcerated on the surface, and projected into the lowermost part of the trachea near the carina.

It is now clear that the tumor, in view of its considerable size, would have to be considered as primary, and the marked lung changes as well as the tumor present in the larynx as metastases. The necropsy confirmed the clinical diagnosis in all points.

Fig. 9 is a representation of the postmortem specimen. The right bronchus shows the primary tumor; above it the narrowed entrance into the left bronchus. Carcinomatous bronchial glands are visible to the right and left of the main bronchi; the right lung appears infiltrated by numerous confluent metastatic tumors, while the cervical glands, the most important drainage region of the larynx, proved to be normal. Consequently the small cancer in the subglottic region of the larynx must be considered as metastatic and not as the primary focus.

Again, this is an instructive example in which only the collective contemplation of the disease-picture permits of the proper use of the various examinations. Those of the specialists were not sufficient; they had to be fitted logically into the clinical picture and evaluated.

On the other hand, another case will prove how effectively the diagnosis and therapy of a case of illness may turn out, if the separate findings fit in logically with one another.

A child, age 1½ years, was brought to the clinic with the statement that six hours previously it suddenly had a chocking attack. By external examination, there was observed slightly accelerated breathing, without stridor. The supplementary roentgen picture (Fig. 10) showed atelectasis of the entire right lung, mediastinal movement with respiration, and the trachea and heart displaced to the right. This pointed to the complete impenetrability of the right bronchus.

Although a shadow-forming foreign body could not be substantiated, the suspicion of a foreign body blocking the right bronchus was nevertheless highly probable. Superior bronchoscopy made possible the perception of a swollen bean in the right bronchus. Five minutes later, after extraction, a new X-ray picture showed almost complete clearing of the right lung with the heart and trachea in the normal position (Fig. 11).

Is it really necessary to go further into the relation of our specialty to general medicine? Are not expressions of disease in the upper air passages only too often the first sign of a general infection? Is not paralysis of the recurrent laryngeal nerve often the first sign of disease of the mediastinal organs and headache the most prominent sign of an accessory sinus affection? A lecture limited by time unfortunately prohibits my illustrating these internal relations to general medicine to as great an extent as is desirable. All these examples would show unequivocally that laryngology and rhinology can be of real worth only as a part of general medicine.

If I now come to speak of surgery, then the mastery of general surgery in our specialty must be looked upon as indispensable. The first generation of laryngologists limited themselves to the endolaryngeal methods, and surrendered every external operation to surgery. Only with difficulty and with the exercise of the most extreme energy has the present generation gradually worked its way into surgical methods. Today, the faultless training in general surgery is a *sine quâ non* for the development of reliable laryngo-rhinologists, masters of their realm.

Only by his training is it possible to think out and carry out faultlessly operation methods, which take into account the individual nature of each case. Only the laryngeal surgeon, who is at the same time master of all the methods of examination of the upper air passages, is best equipped for the development of an individual operative plan.

As an example, I wish to mention the operation for the pharyngeal abscesses and burrowing abscesses of the larynx, cases which as seen externally do not give an indication for an external operation. And yet these abscesses can be controlled only from the outside, after exposure of the vessel sheaths, and by advancing into the parapharyngeal space. If one should wait in these cases until the external examination discloses evident signs of such an abscess, one delays too long. Only the precise laryngoscopic diagnosis coupled with surgical experi-

ence makes possible the right procedure in these cases. To my regret, I must refrain from citing instructive cases in this connection.

*Conclusions:* If the deductions from what has been exhibited are valid, then quite clear conclusions follow.

The specialty of laryngo-rhinology is only conceivable as a useful department in the whole general field of medicine. It will not suffice that young physicians should dedicate themselves to laryngo-rhinology immediately or soon after the completion of their studies. Particularly impracticable is this method for those who are determined to enter upon an academic career and to busy themselves as teachers in the province of medicine. Postgraduate education lasting for years in most of the departments of general medicine and surgery, and a practical knowledge of histology, hemotology and roentgenology, are absolutely requisite as a basis.

We must first be good doctors; only then can we practically and scientifically further develop a specialty, laryngo-rhinology included. In no other way, for a house is not built from the gables toward the foundation stone, but reversed, and the tree bears fruit only when it is fully developed.

Let us, therefore, cherish and protect most carefully the tree of general medicine, so that the fruit of the special department may grow so much the riper and more perfect.

## BRONCHOSCOPY—ITS RELATION TO THE OTHER DEPARTMENTS OF THE HOSPITAL.*

DR. EDWARD KING, Cincinnati.

During the past 30 years, since Killian demonstrated the first successful bronchoscopy, this science has been advancing steadily. At the meeting of the American Laryngological Association in June, V. Mueller and Company, of Chicago, placed on exhibition a very complete and interesting display of all the instruments which have been employed in bronchoscopy from Killian's time until now. This exhibit demonstrated the progressive steps of the specialty. To no man are we indebted more for the advance in bronchoscopy than to Chevalier Jackson. He has brought the subject to its present state of perfection. So great are his accomplishments in this field that he has been recognized and honored throughout the world. During the last meeting of the American College of Surgeons, the Boston Surgical Society took occasion to confer on Dr. Jackson the Henry Jacob Bigelow Medal, an award which has been granted to only three surgeons previously, *viz:* Dr. W. W. Keen, Dr. Wm. Mayo and Dr. R. Matas. In accepting this high honor, Dr. Jackson reviewed the subject of bronchoscopy, stating that over 150,000 bronchoscopies had been performed in this country up to the present time.

My purpose in presenting this subject tonight is to direct your attention to some of the advantages of bronchoscopy and to foster a spirit of co-operation between those who are interested in this subject and the other departments of the hospital.

It is well known that bronchoscopy is indicated in the presence of a foreign body. However, there are other conditions in which it may be employed to advantage: Diseases in which obscure signs may be rendered more clear and very often treatment efficaciously administered.

May I begin by stating that bronchoscopy in the hands of experienced operators is not a difficult nor a dangerous procedure? It is generally performed under local anesthesia, and in young children without any anesthesia. It is not necessary to keep the patient in the hospital, because the procedure requires very little time and there is practically no shock attending it. As a rule we give morphin and

*Read at Staff Meeting of the Good Samaritan Hospital, Nov. 21, 1928.
Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication Dec. 4, 1928.

atropin beforehand, and see that the patient has an empty stomach. In cases of lung abscess and bronchiectasis, the lung is emptied by postural drainage. X-ray examinations are essential and the advice and aid of the radiologist is sought constantly. As I have said before, everyone is agreed that bronchoscopy is indicated when a foreign body is present. As a rule the history is sufficient. The mother states that the child suddenly choked while playing with the object inhaled or swallowed. These are easy to diagnose and examination must be carried out at once. Of course, X-ray plates must be made before the bronchoscopy is attempted. If the object is opaque, accurate information will be obtained as to its size, shape and position. It must be remembered that the X-ray must be made immediately prior to operation, as the foreign bodies very often migrate from one lung to the other, or from one position to another in the esophagus. In cases in which the foreign bodies are not opaque to X-rays, we are aided by physical signs and by X-ray evidence of partial or complete obstruction. But even when no signs are present, and we suspect a foreign body, it is safer to do a bronchoscopy. The non-opaque foreign bodies generally consist of vegetable material, such as beans, seeds or nuts. They are very irritating to the lung and produce severe reactions, with enormous amounts of secretion. Sometimes, unfortunately, the history of aspiration is lacking and the symptoms so vague that the foreign body is overlooked. The symptoms may be those of chronic bronchitis, asthma, lung abscess, bronchiectasis or tuberculosis. Jackson has over 200 cases of overlooked foreign body. These are metal foreign bodies which have been overlooked for periods ranging from six months to 40 years. As a rule, when a history of these cases is obtained, the symptoms date from childhood, when the foreign body entered. Unresolved pneumonia, asthma, etc., are the symptoms, since the foreign bodies are always opaque. In these cases it means that the cause of the trouble might have been discovered had careful X-ray examination been made.

As a rule, any object which will pass through the upper end of the esophagus will pass through the intestines. At times pins, coins, sharp bones and other objects stick in the esophagus and must be removed carefully with the esophagoscope. Blindly pushing a bougie or stomach tube down onto these sharp objects is a dangerous procedure, which may result in perforation.

Here it is important to know the size and shape and position of the foreign body in order that it may be approached with care. In very many of these cases the sharp edges have already perforated the esophagus and there is periesophageal swelling and inflammation,

which can be shown on X-ray. At times, the perforation is disclosed also by X-ray by means of air bubbles which form in the periesophageal tissues at the site of the perforation.

Lung abscess is commanding a great deal of attention at the present time. Probably it is more common. At any rate, the diagnosis and treatment of lung suppuration have received a great impetus in the past few years. It is very often difficult to distinguish between true lung abscess and bronchiectasis, and yet the prognosis of lung abscess is more favorable, because, strictly speaking, lung abscess means a suppuration in the paryenchyma of the lung, while bronchiectatis abscess means suppuration in the bronchi. Thorough evacuation of lung abscess allows the walls to fall together, while the stiff bronchial walls will not collapse and close off the cavity in bronchiectasis. Without considering the treatment of lung abscess, it is surely correct to say that a little more information may be obtained, in addition to X-ray and physical examination, by means of the bronchoscope. No matter where it is located, a direct view will add to our knowledge of the size of the cavity, its contents, and whether there is a foreign body present or a tumor blocking the outlet. Primarily, lung abscess is a disease to be handled by the internist. The patient's general state must be studied and supported. It is he who must determine what measures to pursue in bringing about a cure. The bronchoscopist and the surgeon must have the full co-operation of the internist in order to get results. A certain number of cases must have surgical intervention, and with the rapid advances in thoracic surgery there should be no hesitancy in placing these cases under a surgeon's care. It is incumbent upon us all to study these cases together in order that we may determine as early as possible which cases should be treated medically, which with the bronchoscope, and which by surgery. It will not avail us anything to try all three, for then the case goes to the surgeon so far advanced that the risk is poor. We must learn which method to pursue and adopt that procedure as early as possible.

There is no doubt that the bronchoscope has been of value in the treatment of these cases. Moore, of Philadelphia, has recently reported the results of the treatment of 85 cases, with 46 per cent cured and 20 per cent greatly improved. Moersch, of the Mayo Clinic, in a recent number of *Surgery, Gynecology and Obstetrics*, reports the results of the treatment of 19 cases, in which 16 of this number were cured. My experience teaches me that we can cure with the bronchoscope, centrally located abscesses. The majority of abscesses are probably the result of aspiration, and isn't it logical to

attempt the removal of the infection through the same tract through which it entered? These abscess cavities can be cleaned thoroughly by means of suction, irrigated if necessary, and medication can be applied directly to the walls without any danger to the patient.

The treatment of bronchiectasis is not so encouraging, for the reasons I have given. However, with the infection present there is very often accompanying bronchitis and asthma, and the simple cleansing of the surfaces of the bronchial mucosa with suction and irrigation will very often relieve these symptoms. One of our local men last year read a paper before the Academy in which he reported excellent results with irrigation in lung suppurations. In discussing his paper I reported some cases in which the method had been employed. One of these cases had startling results from the irrigation, and others had indifferent or no results. My series of cases in which this method has been employed is limited to about 10. Some of these patients have had a large number of irrigations, so that my experience with the method is extensive. Perhaps with a larger series of cases our results will be better. Here, again, it is important to determine what case is suitable for irrigation and suction, and by working in close co-operation with the internist, we can select those cases in which the method is calculated to help and eliminate those in which it might do damage. In other words, this Department of Bronchoscopy should be at the call of the Chest Department at all times. Results of treatments should be carefully checked in order that, as I have said before, we may determine the actual value of and the actual indications for this type of treatment.

Recently a report from Jackson's Clinic described the results of bronchoscopy in massive collapse of the lung. The cases were postoperative and the examination revealed a thick plug of mucus blocking the entire lumen of one bronchus. The removal of this plug by suction allowed air to enter the lung at once. My own experience in lung collapse is confined to a case of diphtheria, in which a large membrane obstructed the larynx, requiring tracheotomy. A few days later the left lung collapsed. At the suggestion of the pediatric service, I inserted a bronchoscope and removed a large inspissated mass of membrane. Air entered the lung immediately. It was necessary to repeat this procedure again in a few days. There is a definite place for bronchoscopy in collapse of the lung, especially when we find that the thick mucoid secretion which is causing the obstruction will not move up nor down, no matter in what position the patient is placed. This mass is generally rubber-like in consistency, and when placed in an inverted tube will not flow in either direction.

Recently we saw in our office a patient, age 55 years, whose chief complaint was dyspnea. The patient was a well nourished white woman, who gave the following history: For the past four or five months she had been having increased difficulty in breathing. For the past three weeks she had been in a hospital for diagnosis and observation. A thorough physical examination had been given her. A tonsillectomy had been performed and a large number of teeth extracted. The X-rays were negative. A diagnosis of asthma or hysteria was made. Only on the insistence of the radiologist was bronchoscopy considered by the attending physicians.

On examination the dyspnea was so marked that the patient was unable to lie down. There was retraction of supra and infrasternal spaces. The patient made a loud stertorous noise on inspiration. Bronchoscopy revealed at once the cause of the dyspnea: a tumor protruding from the post wall of the trachea and almost occluding the entire lumen. A large section was removed readily, which proved to be squamous cell epithelioma. The tumor had already invaded the esophagus and glands of the neck, so that it was regarded as hopeless to subject her to surgery.

That cancer of the lung is increasing is proven by literature, by clinical observations, and by autopsies. McCrae, professor of medicine at Jefferson, has pointed out in a recent article that primary cancer of the lung more often involves the bronchi, the lung tissue being secondarily involved. In a combined report by McCrae, Funk and Jackson, 14 cases are presented in which the diagnosis was made by bronchoscopy and corroborated by histologic study. In a study of this report it is noteworthy that the clinical picture varied so greatly that a definite set of symptoms could not be ascribed to this disease. McCrae considers that bronchoscopy is the method of diagnosis which is most certain, if early diagnosis is to be established. The radiologist may be able to say that there is opacity present or a clouding at one point, but it is not always possible for him to give accurate information. Bronchial carcinoma apparently has a rather low-grade malignancy, therefore it is highly desirable that an early diagnosis be established in these cases, and treatment with deep therapy be started, with greater hopes for a cure, or at least prolongation of life.

Cancer of the esophagus constitutes a distressing chapter in the category of malignant diseases. A few years ago high hopes were held out for the cure of these cases by means of radium, directly applied through the esophagoscope, but these hopes have been dissipated by most unhappy results. Lately, I have been impressed with

the results from the use of platinum implantations in the tumor mass and a few reports coming from the New York Radium Clinic have been favorable. The treatment is expensive, however, and the two cases upon whom I have employed it could not continue with the treatment. Here, again, the early diagnosis of the lesion holds forth a promise for a brighter future through surgery. Resection of the upper end has been accomplished successfully and with present efforts directed to the lower end there is a prospect of obtaining some help for these cases. When we consider that any disease which affects the esophagus has the tendency to narrow its lumen, we realize that dysphagia is probably the first symptom pointing to obstruction. Care in examining the history of the patient is essential in order to elicit this symptom. Cancer of the esophagus is the most common cause of dysphagia, and therefore in any case in which this symptom is present it is important to have a look at the esophagus, if early diagnosis is to be established. This is such a simple procedure that it should not be disregarded in any case.

707 Race Street.

## ENDOSCOPIC TREATMENT OF ESOPHAGEAL SUPPURATION.*

DR. RUDOLPH KRAMER, New York.

Suppurative inflammation of the tissues surrounding the esophagus is a complication most dreaded by the esophagoscopist. It is a complication made possible by the mechanical weakness of the esophageal structures and the lack of resistance of the mediastinal tissues to infection. Up to the present time, periesophageal infections, with a few exceptions of abscesses following foreign bodies, have either been allowed to follow the course of nature or have been attacked surgically by external incision and drainage.

The following cases are reported in order to illustrate a plan of attack by way of the esophageal lumen:

*Case 1:* S. I., male, age 49 years, a diabetic, seen on Jan. 7, 1924. The patient stated that three days previously he had swallowed a fish bone. He had pain in both sides of the neck and occasionally in the chest. Slight edema of the uvula was found, with two small ulcerations on its posterior aspect. In the esophagoscope a perforation of the left wall of the esophagus was seen at a distance of 18 c.m. from the upper teeth, from which 5 c.c. of thick, creamy pus was expressed. Lying flat against the posterior wall was a translucent fish bone, 1¼ inches in length, its right end buried in the right esophageal wall, its left end, which was pointed, engaged in the left wall. The lower lip of the perforation of the left wall of the esophagus was punched away to allow better drainage. The day following operation, patient felt well, except for some soreness in the throat. The sharp retrosternal pain disappeared in three days. Six months later the patient stated that he was entirely well.

*Case 2:* Male, age 49 years, seen on Aug. 8, 1925. The patient gave a history of having swallowed a fish bone 10 days previously. He was esophagoscoped the next day, but no foreign body was found. At that time an X-ray showed an obstruction at the cricoid region, interpreted as a malignant growth. Following esophagoscopy, the difficulty in swallowing increased, the patient felt sick and had slight fever. On the following day I was called to see him. In the esopha-

---

*Candidate's Thesis accepted by American Bronchoscopic Society, May, 1928.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication Nov. 16, 1928.

goscope an area of granulation tissue was found at a distance of 16 c.m. from the upper teeth. It was about 1 c.m. in diameter, with an area of necrosis in the center. Upon making pressure with the end of the èsophagoscope, pus was expressed from a pinpoint area in the necrotic portion. The opening was dilated and about 2 ounces of thick, light brown pus escaped. The necrotic tissue forming the margin of the perforation was removed with punch forceps and the cavity aspirated until all of the pus was evacuated. No foreign body was seen. The temperature on the day of operation was 103.2°. In 24 hours it fell to 100° and the patient was able to swallow fluids. Two days later the patient's temperature was normal and complete convalescence ensued. He was well when seen several months later.

*Case 3:* Male, age 53 years. This patient had been complaining of "stomach trouble" for about eight months. At one of the hospitals where he had gone for treatment an esophagoscope was passed under local anesthesia. During this procedure he suffered a great deal of pain, so that the tube could not be passed. The following day he had fever, swelling of the left side of the neck and difficulty in swallowing. On the next day these symptoms became worse, he was unable to swallow anything, and his temperature rose to 103°. At this time I was called in to see the patient. I found a tender swelling on the left side of the neck, extending from the hyoid to the clavicle and from the posterior margin of the left sternomastoid to the right side of the trachea. The trachea was displaced to the right, the pharynx was edematous and there was a bulging on the left side. There was some exudate, with old blood, on the posterior wall of the hypopharynx behind the left arytenoid. The patient was taken to Mt. Sinai Hospital and a consultation was held with the surgeons to consider the advisability of immediate external operation. In view of my experiences with the two cases quoted above, I decided to investigate the possibility of endoscopic treatment before proceeding with the external operation. Upon esophagoscopy, a tear was found on the left side of the hypopharynx just above the cricopharyngeus, running downwards and forwards for a distance of 1 inch, to the anterior wall of the esophagus. The margins of the wound were necrotic and when they were separated, foul pus was evacuated. Esophagoscope was passed into the wound and pus aspirated. The necrotic edges were removed with punch forceps. The esophagoscope was then passed into the lumen of the esophagus. There was a marked edema down to the crossing of the left bronchus, but no other lesion was found. Following the operation there was a marked improvement of all the symptoms, the swelling diminished, the tem-

perature gradually came down, and the patient expectorated foul-smelling pus for several days. During this time he received a rectal drip, but no other feeding. The temperature was normal in four days. The external swelling was markedly diminished, so that the administration of fluids by mouth was begun. At the end of a week there was still some induration in the region of the cricoid and thyroid on the left side. A lymph gland in the left supraclavicular area was palpable. At the end of three weeks the patient was discharged from the hospital, entirely free from symptoms, except for the presence of the supraclavicular gland.

The X-ray examinations during the patient's illness are of interest. At the time of admission to the hospital there was a shadow extending from the root of the neck down to the arch of the aorta. The trachea was displaced to the right. There was probably an exudate in the superior mediastinum pushing the trachea over. One week later there was a decrease in the size of the shadow at the root of the neck. Five days later the barium meal no longer showed any esophageal obstruction, but some of the barium entered a small pocket, apparently on the posterior wall of the esophagus, near the left border and just above the aortic arch. At this time an X-ray of the stomach revealed a defect on the lesser curvature, which was interpreted as a penetrating ulcer. At the end of four months the patient was entirely free from the esophageal trouble, but his stomach complaint had become more pronounced and the X-ray diagnosis was then made of carcinoma of the stomach.

The first case represents a mild form of lesion located within the layers of the esophageal wall, the pus having found its way between the submucous and muscular layers. The periesophageal tissues were only slightly involved.

The second case represents a more serious type of infection. Here we find a necrosis of the superficial layers of the esophageal wall, with more extensive suppuration in the deeper layers, probably involving the deep cervical or periesophageal regions with fairly severe local and constitutional symptoms. In this patient, as in the first, an attempt to facilitate drainage of the abscess and hasten cure was made by removing necrotic tissue and leaving a fairly extensive opening.

These two cases are similar to others reported in the literature and which shall be reviewed briefly later.

The third case is unique. It represents the severest type of esophageal infection, or rather, mediastinal infection following instrumentation during esophagascopy, and as far as I can find from the litera-

ture it is the first case of mediastinal abscess successfully treated by endoscopic methods alone.

Esophageal suppuration may occur as a result of: *1.* Following the use of instruments, either by accidental perforation or following biopsy. *2.* Injury due to the presence of a foreign body. *3.* Stab and gunshot wounds. *4.* Acute esophagitis, as for example, following chemical injury. *5.* Diverticulitis. *6.* Simple or peptic ulcer. *7.* Lues. *8.* Tuberculosis. *9.* Neoplasm.

Foreign bodies, instrumentation and neoplasms are the most common causes. The pus may form superficially beneath the mucosa, but may extend through and between the various layers of the esophagus, finally involving the mediastinum. In fatal cases, thrombosis of the regional veins, pleura, lung or pericardium may occur. (I saw one fatal case of thrombosis of the Vena Azygos).

If the infection is slight there may be no symptoms until spontaneous rupture takes place, when fetid breath and expectoration of foul pus is noted. In more extensive lesions there is pain and tenderness in the neck, followed by swelling along the course of the esophagus. If the swelling is unilateral there is flexion of the head and rotation of the chin to the opposite side. Pulse and temperature are usually proportionate to the severity of the infection. When the lesion is in the region of the cricopharyngeus, changes in the voice and dyspnea occur. Severe toxemia occurs only in the most grave cases. Cases resulting from instrumentation are characteristic. There is a history of severe pain during the instrumentation and the feeling of something "giving way" during the progress of the tube. This is followed by a stormy course of severe mediastinal involvement. Cases following the removal of tissue for biopsy in carcinoma usually start silently with progressive, increasing toxic symptoms, but in cases of perforation following the use of the bougie in carcinoma, the history is the same as in other cases of instrumental perforation. The esophageal findings are as follows: If the lesion is high in the neck there is edema of the arytenoids and swelling of the posterior pharyngeal wall. Lower down, the esophageal lumen is partly or entirely obliterated by edematous swelling of its walls. This may extend throughout the entire length of the esophagus. Distinct evidences of abrasion or perforation may be found, such as granulation, necrosis of a limited area, or the escape of pus from one or more small openings. The foul odor of the pus can usually be recognized during the esophagoscopy. The pus may be creamy, bloody or fetid and thin.

Radiographic findings may reveal a broadening of prevertebral shadows with areas of air or fluid level, or a broadening of the mediastinum at the base of the neck. A lateral picture of the neck region should always be obtained in order to determine the prevertebral thickening and areas of rarefaction due to the presence of air in the tissues.

The prognosis is grave in the extensive lesions. The milder cases localized to the mucous membrane or submucous layers are comparatively benign and usually heal promptly after the removal of any foreign body that may be present and drainage of the abscess. The more extensive periesophageal or mediastinal lesions are extremely serious. External opening of the mediastinum by posterior mediastinotomy is a severe operation, whose prognosis is all the more serious because the cases occur in patients with carcinoma or other severe lesions, or, in very old people or in infants.

The treatment should always begin with esophagoscopy in order to determine the nature of the lesion present, to remove a foreign body, and to evacuate the abscess cavity. General supportive treatment and rectal feeding (fluids) are of course necessary. If improvement does not promptly follow, external drainage should be considered.

The authors who have reported periesophageal cases in the literature are generally in favor of external drainage. Killian advises external operation when there is perforation, emphysema, fever, pain, tenderness, infection and swelling (due to foreign body or trauma), hemorrhage, foreign bodies that cannot be removed, and tracheoesophageal fistulae. Cases that have recovered after external operation have been reported by Iglauer and Ransahoff, Maclay, Haslinger, Seiffert, Roth and Suchanek. Cases that have recovered spontaneously have been reported by Friedenwald and Morrison. Cases in which endoscopic treatment has been followed by recovery, such as in the first two cases reported above, have been reported by Guisez. Of eight cases reported by him, only one died, and this patient was brought to him in extremis. He reports that the patients who recovered showed no ill effects, particularly as regards stenosis, even where the involvement was severe. Seiffert reported a case in 1925. Tucker, in the same year, reported a case following the swallowing of a fish bone, with a hypopharyngeal lesion, which was successfully treated endoscopically.

The successful outcome of the third case reported above encourages me to believe that this form of treatment, which is less formidable, causes less shock, and is liable to fewer complications than the external operation should be employed in all cases of mediastinal suppuration before external operation is considered.

BIBLIOGRAPHY.

SUCHANEK: Wien. Med. Wochen., 1925, LXXV, 470.

TUCKER: *Jour. Amer. Med. Assn.*, 1925, LXXXIV, 511.

ROTH: Zeitsch. f. Hals. Nas. Ohr., 1923, V, 58.

MACLAY: *Jour. Laryn.*, 1923, XXXVIII, 518.

VAN EICKEN: Zeitsch. f. Hals. Nas. Ohr., 1926, XV, 387.

SEIFFERT: Zeitsch. f. Hals. Nas. Ohr., 1925, XII, 290.

GRAVIER and GRIVET: Lyons Med., 1926, CXXXVII, 362.

HASLINGER: Wien. Med. Wochen., 1926, LXXVII, 325.

ORTON: THE LARYNGOSCOPE, 1926, XXXVI, 183.

GUISEZ: Retrecissements de l'Oesophage., Paris, 1923.

GUISEZ: Ann. Mal. d'Oreille, 1924, XLIII, 1041.

GUISEZ: Bull. O., R., L., 1924, XXII, 221.

MCGINNIS: THE LARYNGOSCOPE, 1924, XXXIV, 831.

FRIEDENWALD and MORRISON: *Jour. Amer. Med. Sci.*, 1924, CLXVII, 194.

BALLIN and SALTZSTEIN: Surg. Gyn. and Ob., Jan., 1922, XXXIV, 42.

BROWN, T. A.: Lancet 2, Dec. 16, 1922, 1272.

GUISEZ: Press. Med., April 29, 1922, XXX, 365.

IGLAUER: THE LARYNGOSCOPE, 1924, XXXIV, 821.

121 East 60th Street.

## SIMULATED UNILATERAL DEAFNESS.*

DR. GEORGE W. MACKENZIE, Philadelphia.

This is a subject which has interested the writer for many years, but more especially recently since having seen two cases which escaped detection at the hands of recognized experts who saw them earlier.

When deafness is simulated it is usually of one side only, very rarely of both sides. Furthermore, it is usually claimed to be total and not partial. Simulated deafness, therefore, whether it be prompted by hysteria or malingering, is most often unilateral and total.

There are many tests for the detection of simulated unilateral deafness. In fact, there are so many that to describe them all would require more time that is ordinarily allowable for a single paper. The writer will, therefore, limit himself to a brief description of those methods which he has come to accept as the most reliable after an experience covering the study of a fairly large number of cases. However, he finds no single test that so nearly answers the purpose as that devised by Stenger.

It is not well to approach a case of any kind with a preconceived notion as to what is wrong. Instead, one should start with an open mind and allow his conclusions as to the diagnosis to develop gradually, based solely upon the history, together with findings obtained by most painstaking technique.

It is the practice of the writer to begin by taking a history, the fuller the better, as furnished by the one referring the case, by those closely related to the patient and by the patient himself. He should be allowed plenty of freedom to express himself, even to the point of encouraging him with questions aimed at certain details, which this class of patients are usually fond of emphasizing. A case is recalled in which the patient repeated the remark over and over again that those connected with the plant where he suffered a minor injury "never gave me a bandage or nothin', but sent me home". In taking the history of some of these cases, the stories they tell seem quite ludicrous as compared with that told by the average patient. Under such circumstances it is well to keep a straight face and look credulous, the effect of which is encouraging to the patient when he will frequently talk himself into a trap and convict himself. Even after one is satisfied as to the correct diagnosis, it is well not to let the

*Read before the Philadelphia Laryngological Society, Oct. 10, 1928.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication Oct. 26, 1928.

patient know that the fraud has been discovered until the examination has been completed.

Following the history-taking comes the functional hearing and labyrinthine tests, and these should be conducted in a thoroughly comprehensive manner. Last of all, the ear should be examined with the otoscope in order to learn the exact appearance and behavior of the drum membrane.

It is while making the functional tests that one's suspicion is especially aroused as to the true character of the condition present, which prompts him to make those special tests aimed at the detection of simulated deafness.

The symptoms and signs upon which the writer has come to depend will be cited in the order in which they develop in the study of a case.

*Exaggerated Effort to Hear:* In other words, one who is feigning deafness strains much more to hear than the average individual who is truly deaf. His overstraining efforts are truly theatrical. He puts his hand behind his ear, cranes his neck forward and asks the one who is conversing with him to speak louder and to repeat over and over again, in order to impress the examiner with his infirmity; whereas, an individual who is truly deaf is more anxious to hide it. This symptom begins to show itself early, in fact while still taking the history.

*Lack of the Spirit to Co-operate:* This symptom begins to show itself near the end of the history-taking, but develops more fully during the functional testing. It appears that the patient is afraid the examiner is going to detect the fraud while making tests and rather than suffer the embarrassment of being caught, he connives as best his wit will allow him, to hamper the examiner in his efforts at arriving at definite findings. This prompts the manifestation of the next symptom.

*Variability of Findings:* For instance, while making the hearing test for conversational voice it will be observed that the patient hears the same word, spoken with the same loudness, at one time half a meter distance and at another time during the same visit at 6 meters' distance. We find this same inconsistency even more strikingly while making the tuning fork tests. For instance, in making time tests the simulator will hear the same fork at one trial for 20 seconds and another for 70 seconds. His contradictions go so far as to reveal a positive Rinné one minute and a decidedly negative one the next. Such wide variations in findings do not occur with patients suffering from bona fide deafness.

*Hesitating or Delayed Replies:* This symptom is manifested most typically while testing the patient's hearing for conversational voice.

For instance, if the patient has been asked by the examiner to repeat certain words spoken to him, he fails to respond promptly, as the truthful patient does. If you repeat to him the same word several times he will suddenly strike that attitude which is calculated to impress one with the thought that the word had just dawned on him. This symptom, if it were real, would indicate slow cerebration, and not deafness.

*Repeating Incorrectly:* When an individual who is feigning deafness is asked to repeat certain words after the examiner, he not only overstrains to hear, delays his replies, but will repeat words that do not sound at all like those given him. The actually deaf patient, on the other hand, when he does not hear the words plainly enough to recognize them, calls off promptly words that sound very much like those given him.

*Tinnitus, or Subjective Noises,* is usually absent in this class of cases; at least, it is very rarely complained of as compared with those who are suffering real deafness. The absence of subjective noises, however, should not be taken too seriously in our differential diagnosis, especially since many cases of pronounced deafness are found in which tinnitus is practically absent.

*Vertigo* is usually absent in those feigning deafness. When it is complained of it lacks its accompanying sign, spontaneous nystagmus when looking straight ahead. In those few cases in which the feigners have the temerity to risk complaining of vertigo, they generally fall down in their description of the symptom. For instance, they will often refer to the sensation as that of falling backward, while the true falling reaction of aural vertigo is in the frontal and not the sagittal plane. Again, when referring to the vertigo, they will exaggerate, even to the extent of claiming that they fall down and lose consciousness. Unconsciousness is not a symptom of aural vertigo, but of some intracranial condition, more often cerebral anemia.

*Absence of bone conduction over the mastoid of the deaf side* is a symptom characteristic of unilateral simulated deafness. The contrary is true in the case of bona fide deafness. In the case of simulated deafness the patient is unaware of the presence of positive hearing to bone conduction with a tuning fork applied to any part of the skull, including the mastoid of the deaf side. He, therefore, falls into the error of claiming that he does not hear the fork when applied to the mastoid of the side which he pretends is deaf. A patient who is actually deaf on one side is able to hear the tuning fork when applied to the mastoid of that side, providing there is a fair amount of hearing in the other ear. Of course, he does not hear the fork by bone conduction as loudly over the mastoid of the deaf side as he

does through the bone in the midline of the skull, or through the mastoid of the hearing ear.

*Absence of hearing by Weber test when the meatus of the good ear is closed with the finger:* An individual who is truly deaf, for instance in the right ear, hears the fork when placed on the midline of the skull, and refers the sound of the fork to the hearing side. If the canal of the hearing side is then closed with a finger, he hears the fork more loudly than with the finger out of the ear, because of the improved bone conduction. On the contrary, one who is feigning deafness will usually claim that he ceases to hear the fork altogether when the canal on the hearing side is stopped. Such an answer commits the patient as a cheat.

*Failure of the patient to speak unusually loud when he is made artificially deaf in the hearing ear with a noise producer:* An individual who is afflicted with bilateral nerve deafness speaks appreciably louder than the normal. When a normal individual is made deaf experimentally with two noise producers he will speak unusually loud, like one afflicted with bilateral nerve deafness. Furthermore, an individual who is suffering from total unilateral deafness, for instance, on the right side, will speak as loud and no louder than a normal-hearing individual; but, when his second or hearing ear is made artificially deaf with a noise producer, he will speak louder than normal because of bilateral deafness. On the other hand, one who is feigning deafness on the right side, when he is made artificially deaf on the left side with the noise producer, will continue to talk normally loud because the positive hearing on the right side prevents the occurrence of bilateral deafness. A further proof of the fact that he is feigning deafness is evident the moment we introduce the second noise producer into the right ear, when he speaks more loudly, as happens in all cases of bilateral deafness, whether it be experimentally produced or due to disease.

*Stenger test is negative for true deafness or positive for malingering:* When a normal individual is listening for the source of a sound he lateralizes its direction to the side where he hears it louder.

When the intensity of the sound is equal on the two sides he feels that he is enveloped in the sound and is unable to tell the direction from which it comes.

When the intensity of the sound from two sources is stronger on one side than on the other, the louder sound only is heard. In other words, the fainter sound is drowned out, so to speak, by the louder one. The intensity of the sound can be increased by a stronger stimulation of the fork or by shortening the distance between the fork and the listening ear.

If two forks are used, for instance the double $a_1$ fork of Bezold-Edelmann, which we prefer for the Stenger test, and stimulated alike and one is held much nearer the right ear than the other is to the left, then the right fork is heard quite well, whereas the left one is not heard at all. This of course is true for normal-hearing individuals only. In the case of total deafness, for instance, of the right ear, the left fork is heard while the right one is not, no matter how close the fork on the right side is brought to the ear. In other words, in the case of true unilateral right-sided deafness, no source of sound limited to the right ear can influence the hearing of the left ear. When a disturbance of hearing results from a sound thrown into the right ear, the patient cannot be deaf on the right side.

About four weeks ago Mr. Royce, of the Graybar Electric Company of Philadelphia, brought to the writer's attention a new appliance devised by the Western Electric Company, known as the 42A Test Set. He claimed that it was recommended for use in the detection of malingerers. After a few minutes' conference with Mr. Royce, the writer saw the possibility of the apparatus as a substitute for the double $a_1$ forks of Bezold-Edelmann as a unilateral test for total deafness, with at least two advantages in favor of the instrument, and they are: A constant stimulation, which is not possible with the forks, and a stimulation of equal intensity on the two sides, which is quite difficult of attainment in the case of forks that require stimulation by striking one of the prongs. On the other hand, with experiments thus far conducted it appears that one of the advantages of forks has not yet been met in the new instrument, and that is the high intensity at short range with low carrying power. In other words, the fork, though quite loud at close range, can hardly be heard more than 8 inches from the ear, which prevents any possibility of the sound being carried from one ear over to the opposite one. It is possible that the electrical apparatus might be made to meet this requirement. The technique for making the Stenger test and the results thus far obtained were the same when the tests were made with the electrical instrument as with the forks. It is conceivable at this writing that the advantage of the former may in the long run outweigh those of the latter.

From what has already been said one can see that if a patient reports with the history of total deafness in the right ear following an injury, the otologist ought to be able to determine whether the case is one of bona fide or simulated deafness. Care must be exercised in the study of the case from the very beginning of the history-taking down to the final and most important test, namely, the Stenger test.

In making this test we begin by presenting a vibrating fork to the hearing side, let us say the left, 4 inches from the meatus, when the patient acknowledges hearing it; allowing it to remain there, we next present a second fork to the right side, 2 inches from the meatus. If he fails to hear the one on the left side, it is because it is positive hearing on the right side which interferes with the hearing on the left. If, on the other hand, he continues to hear the fork on the left side it is because he is unquestionably deaf on the right side. This test should be repeated many times before one is warranted in rendering an absolute decision as to the diagnosis, for it is possible that a malingerer may occasionally guess correctly. It is well, too, to try the same test, using a different technique; for instance, after the patient has come to learn that you are using two forks he is prone to guess that he hears the fork on the left side whenever he sees it presented to that side, trying at the same time to ignore the sound of the nearer and therefore louder fork on the right side. He has reached the point where he relies more on what he sees than on what he hears, especially since the fork presented to the right ear is preventing him from hearing the more distant one on the left side. Under these circumstances it is an easy matter to stimulate the two forks with the patient's eyes open and looking at you and then still the fork presented to the left side with the index finger without the patient's knowledge; when he will claim to hear the stilled fork simply because he saw you stimulate it and present it to the left ear. Thus it is that the simulator can be shown to deny hearing a fork when it is vibrating as in the first experiment and of claiming to hear it when it is not vibrating in the second experiment.

*Absence of symptoms and signs referable to the nonacoustic labyrinth:* In the majority of cases of total deafness from injury or disease, there is usually some involvement of the nonacoustic labyrinth, or vestibular nerve, which is not to be found in the case of hysterical or malingering deafness.

If an individual reports with the history of total deafness of sudden onset in the right ear in the absence of spontaneous nystagmus when looking straight ahead, it prompts us to suspect something strange, at least. Furthermore, if the turning test reveals the duration of the after-turning nystagmus to each side to be 24 seconds, our suspicions are strengthened. Finally, if the galvanic test reveals normal figures for the kathodal and anodal reaction on the two sides, there can be no doubt as to the vestibular nerve and the labyrinth being intact and functioning normally.

1724 Spruce Street.

# FOUR CASES OF STREPTOCOCCUS MUCOSIS CAPSULATIS MASTOIDITIS.

Dr. Arthur M. Alden, St. Louis.

The credit for recognizing the fact that mastoid infections due to the capsulated group of streptococci form, from a standpoint of the symptoms exhibited, a distinct otologic entity as compared with the ordinary type of mastoid disease, belongs to Dr. Gohn, professor of pathology at the University of Prague. In 1909, while he was working at the pathological laboratory of the Neumann Clinic, he made the observation that those cases of mastoiditis which in the cultures taken at the time of operation showed either streptococcus mucosis capsulatis or the pneumococcus ran for the most part a course in which the symptoms ordinarily associated with the severe forms of mastoiditis were either very mild or entirely absent. Since this time much attention has been devoted in the European clinics to this group of infections and it is now a well recognized fact that this form of mastoiditis is clinically and pathologically in a class by itself.

The clinical picture of the otitis media due to this infection is much the same as that of any other mild middle ear invasion. The symptoms referable to the middle ear itself are for the most part slight and of relatively short duration and the brunt of the attack in this disease is borne by the mastoid cells. With or without surgical incision of the tympanic membrane, the middle ear manifestations usually subside in a few hours or, at most, in a day or so, while the infection in the mastoid becomes walled off, possibly due to a block in the aditus and a painless and often feverless destructive process slowly takes place. During this stage of the disease, the patient may have no realization whatsoever that anything is wrong with his ear, though several have stated that while they had no pain, the ear did not feel just right, and others have complained of the fact that they did not hear as well as before the onset of the infection. The otologist frequently does not see this type of case until more important symptoms, which in many cases are significant of some grave intracranial complication, become manifest.

The symptoms accompanying the primary infection in the middle ear are often so mild that the patient by this time has even forgotten that he had an ear disturbance until now, when in the presence of

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication Dec. 27, 1928.

impending catastrophe, careful questioning by the otologist makes him recall the fact that weeks or months previous he had a slight pain or sensation of discomfort in the affected ear. In most cases, the acute onset of the disease is so evanescent that the patient does not even consult his family physician until days or weeks later, when the impending intracranial disease makes him realize that something is seriously wrong. Examination of the ear at this time often shows a tympanic membrane that is intact, gray and without bulge, and unless the otologist is conversant with the peculiar symptom-complex of this disease, he may find it hard to convince himself that the original site of infection was in the ear.

The four cases which I shall report all gave pure cultures of streptococcus mucosis capsulatis from the pus taken at the time of operation.

*Case 1:* Mr. J. M., a retired business man, age 64 years, who was seen on account of fever and unconsciousness, accompanied by a discharging left ear. The history obtained from the family was that about two months prior the patient had a mild attack of grippe. Three days following this he had pain in the left ear, which lasted for about 24 hours. On account of this, he consulted another otologist, who opened the eardrum, and the patient had been under his care until a few days before the present attack. The family said that the discharge from the ear during this time had been slight and that the patient had at no time complained of pain in or around the ear. The afternoon before I saw him he was doing some little work in his place of business when suddenly a considerable amount of pus ran out of the ear and down onto his face. Shortly after this he developed a severe headache on the left side and went home. On arriving home he complained of dizziness and was unable to eat his supper. At 8 o'clock his temperature was 103° and he spent a very restless night, at times talking at random, as though unaware of where he was or what had happened to him. I saw the patient at 8 o'clock the following morning. At that time his temperature was 103.6° and he was in a semicomatose condition, from which he could be aroused only with difficulty. His answers to questions were incoherent and his mental faculties were definitely disturbed. His pupils were regular but reaction to light was very sluggish, and to accommodation could not be determined. He had a conjugate deviation to the right. He was sent immediately into the hospital for more detailed examination. On arrival there his blood pressure was 100/35, his RBC was 4,000,000, and WBC, 16,000, with 75 per cent polymorphonuclear leukocytes. There was a small amount of mucoid discharge

in the ear canal, which, when removed, disclosed a nipple perforation in the posterior-superior quadrant of the eardrum. The remainder of the drum was gray and normal in position. The hearing in either ear could not be determined on account of the condition of the patient, though with the Barany alarm apparatus in the right ear he responded to questions directed to the left. The patient was sent to the radiological department for mastoid plates, which showed complete obliteration of all cell detail on the affected side. The other side was a large, normal, pneumatic mastoid. A tentative diagnosis of mastoiditis with probable brain abscess was made and immediate operation decided upon.

The mastoid was opened under gas anesthesia. A very thick cortex was encountered, which, when removed, showed a completely disintegrated mastoid with a small exposure of the dura in the region of the tegmen antri. Removal of some of the bony plate in this region was followed by a gush of thick, yellow pus, which apparently came from underneath the temporal lobe. The dura was exposed widely but was not bulging or tense and no other evidence of brain abscess could be found. The lateral sinus was not thrombosed, though there were granulations over its external wall. Patient came off the operating table at 5:30 p. m., apparently no worse for the operation. At 6:30 p. m. 500 c.c. of glucose solution was given intravenously. This was followed at 8 o'clock by a chill, which lasted about 10 minutes. The patient at no time following operation regained consciousness and his breathing became increasingly more stertorous. At 2 a. m. his temperature was 107° and at 2:55 a. m. he expired. An autopsy was refused by the family.

*Case 2:* V. L. R., a school boy, age 6 years, was brought into the office by his father on account of a swelling behind the right ear. The father said that 10 days ago the boy had complained for a few hours of headache and pain in this ear. This was in the evening and because the following morning the child appeared normal a doctor was not consulted. The boy had gone to school every day since this time but the evening before I saw him he again complained of pain in the ear and the mother noticed that the skin just behind and above the ear was somewhat swollen. The father says that the swelling is much worse than it was the night before. The child's temperature was normal and he was to all appearances not sick. Examination of the ears showed the left drum to be normal and the right to be intact, though lustreless and somewhat reddened in the upper half. Behind the ear was a manifest subperiosteal abscess. There was no nystagmus or vertigo and both pupils were regular and reacted normally to

light and accommodation. It was only with difficulty that the family were convinced that this little patient had a serious type of infection, but after some argument he was sent to the hospital for operation. On arrival at the hospital his WBC was 17,000, with 82 per cent polymorphonuclear leukocytes. Eye ground examination showed the right disc to be somewhat hazy, the left was normal. All other neurologic findings were negative. X-rays showed complete destruction of the right mastoid.

Under gas anesthesia the mastoid was opened and found to be completely filled with pus and granulations. The bone covering the dura of the lateral sinus, the upper part of the cerebellum and the floor of the middle fossa was necrotic. A large perisinus abscess containing about 1 ounce of thick, yellow pus was drained. No evidence of brain abscess could be seen. The dura was covered with rubber dam, the wound lightly packed with gauze and no effort at surgical closure was made. The child went off the table in good condition. Temperature rose to 102° the night following operation but was normal the next day and remained so throughout his convalescence. On the eleventh day following operation a secondary closure of the mastoid wound was done under gas and the child discharged from the hospital two days later. His convalescence from that time was uneventful.

*Case 3:* J. W. C., a young man, age 22 years, was referred on account of pain and swelling behind the left ear. He says that three months ago he was in bed for two days with grippe. A few days after he was up and around he had a sensation of discomfort in the left ear, which lasted for several days. This symptom was not severe enough to cause him to consult a physician and since it disappeared patient has felt well and worked every day, but thinks that his hearing on the affected side is not as good as it was before his illness. Three days ago he was awakened in the morning by pain in the ear and noticed that there seemed to be a soft, swollen spot just behind and above the ear. The pain subsided after a dose of aspirin, but the swelling has become daily more prominent.

Examination showed the right eardrum to be normal and the left to be intact and lustreless, with a slight bulge in the membrana flaccida. There was no sag of the superior canal wall. There was a spot of fluctuation about 1¾ inches above and behind the ear. X-ray showed complete obliteration of all cell detail and a point of distinct rarefaction, at which the external rupture had probably occurred. The X-ray of the unaffected side demonstrated a large normal, pneumatic mastoid. The patient was sent into the hospital for operation.

On arrival at the hospital his temperature was normal, his WBC, 10,400, with 78 per cent polymorphonuclear leukocytes. Neurologic examination was entirely negative.

Under gas anesthesia a typical simple mastoid operation was done. The majority of the intracellular partitions were necrotic, but a few remained in the region of the tip. All cells were filled with mucopus or granulations, or both. The fistula through the external cortex was 1¾ inches above and behind the external auditory canal. Underneath this lay a large abscess cavity containing about 15 c.c. of free pus, in which the dura of both the middle fossa and the lateral sinus lay exposed and covered with granulations. The sinus was not thrombosed. All pneumatic structures were cleaned out, the dura covered with rubber dam and the mastoid wound was closed in its upper half. The patient's temperature did not show a postoperative rise and remained normal throughout his convalescence. He was allowed out of bed on the fifth day following operation and was discharged from the hospital on his eighth postoperative day.

*Case 4:* A. H., boy, age 4 years. On Feb. 15, following a slight rhinitis, the child had earache on the left side, which lasted all one night. The mother thought that he had some fever, but did not know how much. The next morning the pain was gone and he seemed well. Two days later for a few hours he had an earache on the right side, but this also subsided after the administration of home remedies. March 1, following a very uncomfortable night, he was brought to the hospital clinic, complaining of pain in both ears, and upon examination his temperature was 100.2° and both eardrums were lustreless and bulging. Both were incised under ethyl-chlorid and a small amount of mucoid secretion released from each side. He was sent into the hospital for observation and treatment. Two days later both eardrums had closed and remained so until he was discharged on March 8, apparently entirely recovered. The patient re-entered the hospital on March 11 because of fever and pain in the left ear. Examination at this time revealed an apparently normal right eardrum, while the left was dull and retracted. There was slight tenderness over the tip of the mastoid and the temperature was 100°. WBC, 11,000. Because the child had a slight bronchitis and a red throat, his fever was thought to be due probably to the upper respiratory infection, rather than the ear, and the patient was not seen again until March 13, when a marked change in his general appearance was noticed. He looked septic, though his temperature was 99°, WBC was 16,400, RBC, 3,950,000, and hemoglobin, 62 per cent. On examination the left drum presented much the same appearance as

before, dull gray in color and retracted. Tenderness over the mastoid was questionable, but there was a definite spot of tenderness and edema about 1¼ inches behind the external auditory canal and over the exit of the mastoid emissary vein (Griesinger's sign). Immediate X-rays were taken, which demonstrated some cloudiness over the left mastoid as compared with the right, though all of the intracellular partitions appeared to be intact. Based upon the child's history, his manifest sepsis and the Griesinger sign, which is usually indicative of a thrombotic block to the mastoid emissary vein, a diagnosis of left mastoiditis with probable thrombosis of the lateral sinus was made and immediate mastoid operation advised.

Under ether anesthesia the mastoid was opened and a diploetic structure, with some pus in the cells but little bone destruction, revealed. The aditus was very small and completely filled with edematous mucosa. Removal of the bone covering the lateral sinus exposed a small perisinus abscess and a lateral sinus, which was white and completely obliterated. The internal jugular vein was tied in the lower triangle and the lateral sinus opened. It was filled with a firm, white clot, which had begun to soften, in the region just above the jugular bulb. Free bleeding was not obtained from the posterior end, although exposure was carried backward to within one-half inch of the superior longitudinal sinus. The child was sent off the table in only fair condition and was given an immediate transfusion of 350 c.c. of blood. The postoperative reaction was slight and his convalescence was uneventful until March 19, when he developed a rash, which was diagnosed as measles, and the patient was sent to the isolation hospital. On arrival here, the rash was found not to be typical of measles, but more suggestive of a toxic erythema. Five days later the temperature again went up and the child had a manifest bronchopneumonia, from which he succumbed April 1.

537 Frisco Building.

# LESIONS OF THE COCHLEA EXPERIMENTALLY PRODUCED IN GUINEA PIGS BY INJECTING FECAL EXTRACT FROM CASES OF PROGRESSIVE DEAFNESS. PRELIMINARY COMMUNICATION.*

Dr. Mark J. Gottlieb, New York.

This research work was undertaken for the purpose of determining whether or not the excreta of persons who were suffering with a deafness of a progressive character could produce, when injected into guinea pigs, a lesion of the organ of hearing. Naturally, it was necessary to control the experiment by injecting the excreta of persons who had normal hearing into animals for the sake of comparison.

It has been my impression for some time that the cause of progressive deafness was remote from the ears and that poisonous material carried by the circulation produced such changes in the hearing organ as to render it incapable of performing its normal function[1].

Some people believe that the labyrinthine capsule is involved in some peculiar bony change which impairs the function of the contents of that bony casing. Some otologists assume that these structural changes are caused by pathological conditions in the environs of the labyrinthine capsule. Others believe that such changes are due to some unknown metabolic disorder. Kopetzky and Almour[2] have shown recently that abnormal calcium and uric acid figures are encountered in the blood in cases of progressive deafness. It is assumed by others that the gradual destruction of hearing in certain individuals is due to an endocrine disturbance.

Certain it is that on delving carefully into the history of cases of deafness of the type under consideration, one is often struck by the association of other abnormalities, such as constipation, diarrhea, mucous colitis, hay fever, asthma, vasomotor rhinitis, generalized erythema, urticaria, arthritis, psoriasis, lesions of the bones[3], and contraction of the visual form and color fields[4]. These manifestations often begin before the apparent onset of loss of hearing, or they may occur synchronous with the ear symptoms.

The nearest approach to the work herein contained is that of Leicher[5], who says: "The serum, whole blood and, to a certain

*From the Pathological Department of the Hospital for Joint Diseases, New York. This research is subsidized by a philanthropist, who does not desire publicity.

extent, urine, saliva and sweat of otosclerosis patients show a much stronger poisonous effect on plant cells than do similar body fluids from healthy, nonmenstruating subjects. The poisonous effects showed themselves in an early death of cut flowers of equal age and in an inhibiting influence on the fermentation of yeast. The poisonous effect is found also in extracts of alcohol, chloroform, ether and acetone, but is absent in the dialysate. The phosphatids are most probably the carriers of the poison. Otosclerotic patients while menstruating show an increase of the poisonous effect on plants."

The subject-matter of this report deals with the effects on guinea pigs of extracts of stool from four cases of deafness and three healthy individuals in whom no sign of deafness exists. The cases of deafness include three whose hearing difficulty is progressively getting worse, and one whose hearing impairment is stationary. These cases have a few things in common, but differ in certain essential details, which will be found in the histories. None of these patients were menstruating at the time the specimens were obtained.

The feces were obtained in a liquid form after a cathartic containing phenophthalein. Two ounces or more of the liquid stool was beaten up in a large beaker with 400-500 c.c. of normal saline solution, depending upon the consistency of the stool. The saline solution contained 0.25 per cent of phenol. This mixture was placed in the ice chest and allowed to remain at a low temperature for 24 hours. This was stirred once daily for eight to 10 days in succession. It was then centrifuged at high speed until clear. The clear, supernatant fluid was then cultured for sterility. If a growth was obtained, it was sterilized by subjecting it to heat in a water bath at 56° C. for one hour each day for three successive days and again cultured. The extracts were made by the same person in each instance. The concentration of each extract naturally varied, but the amount of variation was felt to be small. No effort was made to determine its chemical constituents. From this standpoint, the result of the investigation must always be open to question. Such crudity was, however, necessary in this first experiment, because we were dealing with unknown quantities and qualities, which will receive careful consideration in future experiments.

When each experiment was finished, the guinea pigs were killed by cutting the throat and allowing them to bleed to death. The animals were not stunned first. Before the bodies had cooled, the temporal bones were removed from the skull, the apex of the cochlea pierced with a fine needle and then immersed in 10 per cent formalin; decalcified either with nitric acid or Miller's solution and mounted in paraffin.

The diet of the guinea pigs was in no way different from that given to non-experimental animals. The injections were given subcutaneously.

The beginning of this research was done at the Lowenthal Pathological Laboratory. It was then transferred to the pathological laboratory of the Hospital for Joint Diseases. Appreciation for the hospitality given by the Hospital for Joint Diseases is herewith acknowledged. Thanks is also extended to Dr. Henry L. Jaffe, director of the pathological laboratory, for his help and guidance. Dr. Jaffe has reviewed all of the pathological material, and his opinion of the results obtained will be found in the pathological reports following each experiment.

*Experiment 1:* Specimen of stool of Miss H. M., age 27 years, single. Difficulty in hearing started five years previously after having a cold in the head and chest. It did not start with tinnitus; no hereditary history. Says she did not have discharge from ears; no vertigo. The hearing of the right ear is impaired more than the left. The drums are dull and retracted. Eustachian tubes patent. Hearing not improved after inflation. The urine, Wassermann and blood chemistry were normal.

HEARING TESTS.

| | *Right* | *Left* | *Normal* |
|---|---|---|---|
| Conversation | 4½ feet | 2 feet | 40 feet |
| Watch | 2 inches | 2 inches | 3 feet |
| A | negative | negative | 22 seconds |
| 256 D. V. | | | |
| B | 15 seconds | 12½ seconds | 15 seconds |
| 26 D. V. | negative | negative | |
| 128 D. V. | negative | negative | |
| 512 D. V. | positive | positive | |
| 1024 D. V. | positive | negative | |
| 2048 D. V. | positive | positive | |
| 15000 D. V. | positive | positive | |
| 20000 D. V. | negative | negative | |
| Weber | referred to left | | |

Extract of stool injected into a medium-sized pig (Laboratory No. 1). Twenty-one injections were given, as follows:

April 4, 1927, 0.5 c.c. April 18, 1927, 1.5 c.c. May 2, 1927, 1.5 c.c.
April 6, 1927, 1.0 cc.. April 20, 1927, 1.5 c.c. May 4, 1927, 2.0 c.c.
April 8, 1927, 1.5 c.c. April 22, 1927, 1.5 c.c. May 6, 1927, 2.0 c.c.
April 10, 1927, 2.0 c.c. April 24, 1927, 1.5 c.c. May 8, 1927, 2.0 c.c.
April 12, 1927, 1.5 c.c. April 26, 1927, 1.5 c.c. May 10, 1927, 2.0 c.c.
April 14, 1927, 1.5 c.c. April 28, 1927, 1.5 c.c. May 12, 1927, 2.0 c.c.
April 16, 1927, 1.5 c.c. April 30, 1927, 1.5 c.c. May 14, 1927, 2.0 c.c.

On May 16, the animal was killed by cutting the throat with sharp scissors. No gross abnormalities were found on autopsy. Both temporal bones were prepared for section. Section normal (Fig. 1).

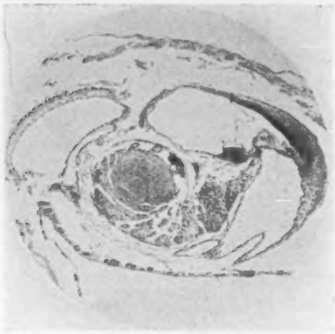


Fig. 1. Experiment 1. Guinea pig No. 1. Section through cochlea normal.

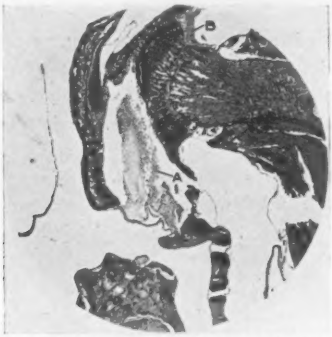


Fig. 2. Experiment 2. Guinea pig No. 2. (A) Old and recent hemorrhage into the vestibule of the cochlea. (B) Old and new hemorrhage into scala tympani.

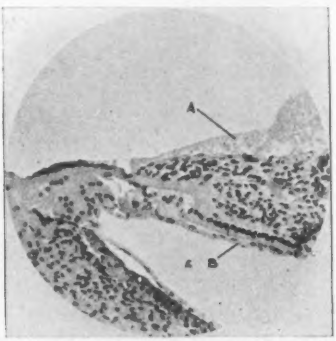



Fig. 3. Experiment 2. Guinea pig No. 2. (A) Recent and old hemorrhage on the under surface of the lamina spiralis and organ of Corti. (B) Membrana vestibularis ruptured and applied to upper surface of organ of Corti (artifact).

*Experiment 2:* Specimen of stool of Miss D. W., age 25 years, single. Impairment of hearing began four years ago, preceded by tinnitus. Since then the hearing has rapidly become markedly reduced. Never had discharging ears or vertigo. No hereditary history. Both drums are normal and Eustachian tubes are patent. Hearing not improved after inflation. The urine is normal, the Wassermann is negative.

BLOOD CHEMISTRY.

| | |
|---|---|
| Uric acid | 1.8 mgms. (normal 1-3 mgms. per 100 c.c.) |
| Calcium | 9.6 mgms. (normal 8-10 mgms. per 100 c.c.) |
| Phosphorous | 3.8 mgms. (normal 2-6 mgms. per 100 c.c.) |

HEARING TESTS.

| | *Right* | *Left* | *Normal* |
|---|---|---|---|
| Conversation | 2 feet | 1 foot | 40 feet |
| Watch | at concha | negative | 3 feet |
| A | 11 seconds | 7 seconds | 22 seconds |
| 256 D. V. B | 18 seconds | 17 seconds | 15 seconds |
| 26 D. V | negative | negative | |
| 128 D. V. | negative | negative | |
| 512 D. V. | positive | positive | |
| 1024 D. V. | positive | positive | |
| 2048 D. V. | positive | positive | |
| 15000 D. V. | positive | positive | |
| 20000 D. V. | negative | negative | |
| Weber | referred to right | | |
| Gellé | negative | positive | |

Result of X-ray of skull, pelvis and long bones by Dr. Henry K. Taylor. Partially sclerotic mastoid processes and bilateral sacro-iliac arthritis.

Extract of stool injected subcutaneously into a medium-sized guinea pig (Laboratory No. 2). Sixteen injections were given, as follows:

April 4, 1927, 0.5 c.c. April 16, 1927, 0.5 c.c. April 28, 1927, 1.0 c.c.
April 6, 1927, 1.0 c.c. April 18, 1927, 1.0 cc.. April 30, 1927, 1.0 c.c.
April 8, 1927, 1.5 c.c. April 20, 1927, 1.5 c.c. May 2, 1927, 1.0 c.c.
April 10, 1927, 2.0 c.c. April 22, 1927, 0.5 c.c. May 4, 1927, 1.0 c.c.
April 12, 1927, 0.5 c.c. April 24, 1927, 1.0 c.c.
April 14, 1927, 1.0 c.c. April 26, 1927, 1.0 c.c.

After each injection the pig was sick. Its hair would stand on end. It would sneeze a good deal and the eyes were constantly moist. At times during the course of the experiment it looked as though the animal had difficulty in orientation.

May 6, the animal was killed by cutting its throat. The autopsy disclosed subperiosteal hemorrhages of the scalp and congestion of both lungs.

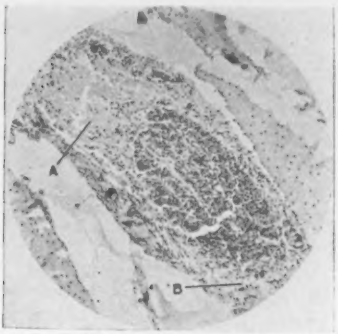



Fig. 4. Experiment 2. Guinea pig No. 2. (A and B) Hemorrhage around geniculate ganglion in the facial nerve.

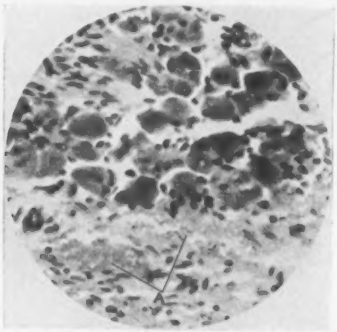



Fig. 5. Experiment 2. Guinea pig No. 2. (A) Red blood cells in facial nerve around geniculate ganglion.


Fig. 6. Experiment 3A. Guinea pig No. 3. (A) Hemorrhage undergoing organization. (B) More recent evidence of bleeding.

The sections showed extensive old and new hemorrhages into the cochlea, hemorrhages into the internal auditory meatus, and around the geniculate ganglion (Figs. 2, 3, 4 and 5).

*Experiment 3:* Stool obtained from Mrs. F. R., age 30 years. Patient has become progressively deaf for the past nine years. Began with tinnitus. Says that the left ear is worse than the right and that the progression of deafness in the right ear had ceased about one year ago. Bowels are constipated. For the past 20 years has had psoriasis. One sister is deaf. Suffers now with occasional attacks of dizziness and vomiting and headaches. The right ear shows an area of redness around the hammer handle. The left external auditory canal is red in its upper portion. The drum is retracted. The patient has a severe generalized psoriasis.

HEARING TESTS.

March 10, 1928.

| | *Right* | *Left* | *Normal* |
|---|---|---|---|
| Conversation | 4 feet | 6 inches | 40 feet |
| Watch | at concha | negative | |
| A | 8 seconds | negative | 22 seconds |
| 256 D. V. | | | |
| B | 12 seconds | 17 seconds | 15 seconds |
| 26 D. V. | negative | negative | |
| 128 D. V. | negative | negative | |
| 512 D. V. | 21 seconds | 15 seconds | 60 seconds |
| 1024 D. V. | 15 seconds | negative | 50 seconds |
| 2048 D. V. | 15 seconds | negative | 30 seconds |
| Weber | not referred | | |

The color and form fields are normal. The Wassermann is negative.

BLOOD CHEMISTRY.

Calcium 7.4 mgms. per 100 c.c. (normal 8-10)

Uric acid 4.9 mgms. per 100 c.c. (normal 1-3 )

The extract from the stool was injected into a guinea pig as follows: *Experiment 3A:* Brown, tan and white guinea pig. Female. Small, Laboratory No. 3:

| | | |
|---|---|---|
| June 23, 1927, 1.0 c.c. | July 7, 1927, 3.0 c.c. | July 25, 1927, 1.0 c.c. |
| June 24, 1927, 2.0 c.c. | July 8, 1927, 1.0 c.c. | July 27, 1927, 1.0 c.c. |
| June 25, 1927, 3.0 c.c. | July 9, 1927, 2.0 c.c. | July 29, 1927, 1.0 c.c. |
| June 27, 1927, 1.0 c.c. | July 11, 1927, 3.0 c.c. | July 30, 1927, 1.0 c.c. |
| June 28, 1927, 2.0 c.c. | July 13, 1927, 1.5 c.c. | Aug. 1, 1927, 1.0 c.c. |
| June 29, 1927, 3.0 c.c. | July 15, 1927, 1.5 c.c. | Aug. 3, 1927, 1.0 c.c. |
| June 30, 1927, 1.0 c.c. | July 16, 1927, 2.0 c.c. | Aug. 5, 1927, 1.0 c.c. |
| July 1, 1927, 2.0 c.c. | July 18, 1927, 2.0 c.c. | Aug. 6, 1927, 1.0 c.c. |
| July 2, 1927, 3.0 c.c. | July 20, 1927, 2.0 c.c. | Aug. 8, 1927, 1.0 c.c. |
| July 5, 1927, 1.0 c.c. | July 22, 1927, 1.0 cc. | Aug. 10, 1927, 1.0 c.c. |
| July 6, 1927, 2.0 c.c. | July 23, 1927, 1.0 c.c. | Aug. 12, 1927, 1.0 c.c. |

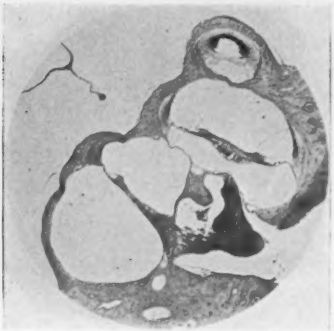


Fig. 7. Experiment 3B. Guinea pig No. 6. Section through the cochlea shows no abnormality.

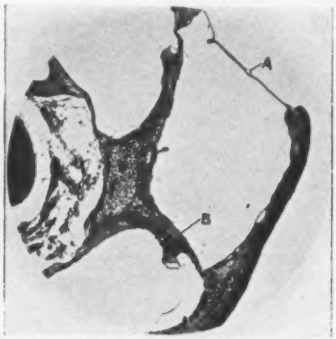



Fig. 8. Experiment 4A. Guinea pig No. 5. (A) Remains of recent bleeding in region of round window on inner surface of the membrane. (B) Organizing hemorrhage.


Fig. 9. Experiment 4A. Guinea pig No. 5. (A) Extensive old and new hemorrhage from inner surface of membrane of round window extending onto under surface of lamina spiralis.

The pig was killed on Aug. 15, 1927. Sections show moderate organized old bleeding into the cochlea (Fig. 6).

*Experiment 3B:* White and black, small guinea pig, female, Laboratory No. 6. Same extract used here as in Experiment 3A.

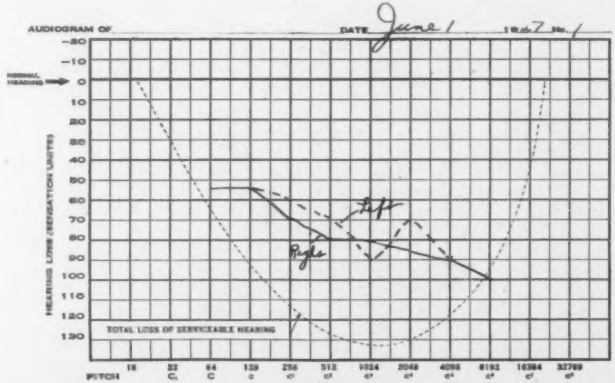
| | | |
|---|---|---|
| July 18, 1927, 2 c.c. | July 27, 1927, 1 c.c. | Aug. 5, 1927, 1 c.c. |
| July 20, 1927, 2 c.c. | July 29, 1927, 1 c.c. | Aug. 6, 1927, 1 c.c. |
| July 22, 1927, 1 c.c. | July 30, 1927, 1 c.c. | Aug. 8, 1927, 1 c.c. |
| July 23, 1927, 1 c.c. | Aug. 1, 1927, 1 c.c. | |
| July 25, 1927, 1 c.c. | Aug. 3, 1927, 1 c.c. | |

The pig was killed on Aug. 9, 1927. Sections show no apparent abnormality (Fig. 7).

*Experiment 4:* Specimen of stool from Mr. I. F., patient of Dr. Samuel J. Kopetzky, age 26 years. June 1, 1927.

*History:* Has had a gradual loss in hearing for past six years. Has had several nasal operations, also inflation and massage, but no relief. No venereal disease. No tinnitus, headache or vertigo. Father and two sisters hard of hearing. No other ailments.

X-ray of skull, long bones and pelvis showed the following: Skull: The cranial vaults appear slightly thicker than the average, but within normal limits. At the upper portion of the occipital bone, there is a small radiolucent area. There is a slight thickening of the inner table, at the posterior portion of the frontal bone. Right mastoid: The tip of the mastoid and major portion of the mastoid proper is composed of diploic structure and is a sclerotic change. There are sclerotic changes in the temporal bone and immediately above the tegmen. Left mastoid: The posterior limit of the mastoid proper shows sclerotic changes.




Bone conduction. Right, 24/20; left, 18/20.

BLOOD CHEMISTRY.

| | |
|---|---|
| Sugar | 90 mgms. per 100 c.c. (normal 60-120) |
| Urea nitrogen | 15 mgms. per 100 c.c. (normal 12-15 ) |
| Uric acid | 3.3 mgms. per 100 c.c. (normal 1-3 ) |
| Calcium | 7.1 mgms. per 100 c.c. (normal 8-10 ) |

*Experiment 4A:* Mr. I. F.

Black, brindle and tan, male guinea pig, small, Laboratory No. 5:

| | | |
|---|---|---|
| June 23, 1927, 1.0 c.c. | July 7, 1927, 3.0 c.c. | July 25, 1927, 1.0 c.c. |
| June 24, 1927, 2.0 c.c. | July 8, 1927, 1.0 c.c. | July 27, 1927, 1.0 c.c. |
| June 25, 1927, 3.0 c.c. | July 9, 1927, 2.0 c.c. | July 29, 1927, 1.0 c.c. |
| June 27, 1927, 1.0 c.c. | July 11, 1927, 3.0 c.c. | July 30, 1927, 1.0 c.c. |
| June 28, 1927, 2.0 c.c. | July 13, 1927, 1.5 c.c. | Aug. 1, 1927, 1.0 c.c. |
| June 29, 1927, 3.0 c.c. | July 15, 1927, 1.5 c.c. | Aug 3, 1927, 1.0 c.c. |
| June 30, 1927, 1.0 c.c. | July 16, 1927, 2.0 c.c. | Aug. 5, 1927, 1.0 c.c. |
| July 1, 1927, 2.0 c.c. | July 18, 1927, 2.0 c.c. | Aug. 6, 1927, 1.0 c.c. |
| July 2, 1927, 3.0 c.c. | July 20, 1927, 2.0 c.c. | Aug. 8, 1927, 1.0 c.c. |
| July 5, 1927, 1.0 c.c. | July 22, 1927, 1.0 c.c. | Aug. 10, 1927, 1.0 c.c. |
| July 6, 1927, 2.0 c.c. | July 23, 1927, 1.0 c.c. | Aug. 12, 1927, 1.0 c.c. |

On Aug. 15, 1927, the pig was killed. At autopsy there was a noticeable hemorrhage under the scalp between and over the eyes.

Sections show old blood pigment at attachment of organ of Corti to ligamentum spirale. On inner side of membrane of round window thick layer of disintegrating red blood cells and fibrin was found and deposit of disintegrating red blood cells and white cells under membrana spiralis ossea in middle coil. Hemorrhage in dura in region of internal auditory meatus (Figs. 8, 9, 10 and 11).

*Experiment 4B:* All-brindle, female guinea pig, small, Laboratory No. 4. Extract of feces of I. F. injected on the following dates:

| | | |
|---|---|---|
| July 18, 1927, 2 c.c. | July 27, 1927, 1 c.c. | Aug. 5, 1927, 1 c.c. |
| July 20, 1927, 2 c.c. | July 29, 1927, 1 c.c. | Aug. 6, 1927, 1 c.c. |
| July 22, 1927, 1 c.c. | July 30, 1927, 1 c.c. | Aug. 8, 1927, 1 c.c. |
| July 23, 1927, 1 c.c. | Aug. 1, 1927, 1 c.c. | |
| July 25, 1927, 1 c.c. | Aug. 3, 1927, 1 c.c. | |

Pig became ill on Sunday, Aug. 7, 1927. Killed on Aug. 8, 1927, four hours after injection. Autopsy negative.

Sections show in vestibule of labyrinth on inner side of membrane of round window, on entire extent of under surface of membrana spiralis and both organs of Corti of basal coil organizing bloody exudate. Old hemorrhage into middle coil of cochlea. Fresh hemorrhage into dura (Figs. 12, 13 and 14).

*Case 1* is undoubtedly one of involvement of the cochlea, due to a local infection. The damage produced at the time of the infection impaired the hearing up to a certain point and then ceased. The hearing of this patient will remain as it is. No lesion was produced

in the guinea pigs by injecting extract of stool from this patient. This stool was not found to be toxic in the doses used.

*Case 2:* Here is a patient whose hearing is rapidly being destroyed. The result of the hearing tests are of the same character as that found in cases diagnosed as otosclerosis. However, the blood chemistry figures come within the range of normal. The extract of stool from the patient produced in small doses definite severe lesion of the cochlea.

*Case 3:* This patient is moderately deaf, has psoriasis, abnormal blood chemistry figures, normal fields for form and color, and obstructed Eustachian tubes. There are psoriatic patches in the external auditory canals and on the drums. At the Eustachian orifices there are areas of polypoid degeneration. The hearing tests are not typical of any one condition. It results probably from a combination of involvement of the cochlea and middle ear. In this case the two conditions are resultant from a common cause. The factor which produces the psoriasis is also the cause of the condition of the ears. The extract of stool from this patient in small doses is not toxic to guinea pigs, but in larger doses definite lesions are produced.

*Case 4* is a case of rapidly advancing deafness. It has all the earmarks of deafness of constitutional origin. The stool extract is toxic to guinea pigs in both small and larger doses.

*Controls:* The controls consist of three guinea pigs that were kept with the experimental animals, but not treated. They were killed during the course of the experiment. They are designated, "Normal Control, Nos. 1, 2 and 3". The "Experimental Controls" are six guinea pigs treated with stool extract from three apparently normal persons and having normal hearing. Two animals were injected with the stool of one individual. The treatment of one is more intense than the other. They are designated, "Experimental Control, No. 1A and B, No. 2A and B, No. 3A and B".

Normal Control, No. 1, Pig, Laboratory No. 225, weight 300 gm. Feb. 25, 1928, killed by cutting throat. Both ears and eyes placed in Miller's solution. No apparent microscopic changes.

Normal Control, No. 2, Pig, Laboratory No. 246, received Feb. 18, 1928; weight 445 gm; April 24, 1928, weight 550 gm. Killed by chloroform and cutting throat; more blood in tissues than by just cutting throat. Eye and both ears in formalin and then Miller's solution. Sections: Fresh blood found in the middle ear, cochlea normal.

Normal Control, No. 3, Pig, Laboratory No. 325, weight 550 gm. April 24, 1928. Killed by cutting throat with scissors. Eyes and ears

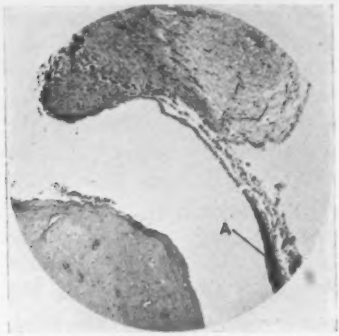



Fig. 10. Experiment 4A. Guinea pig No. 5. (A) Hemorrhage into dura in region of internal auditory meatus.

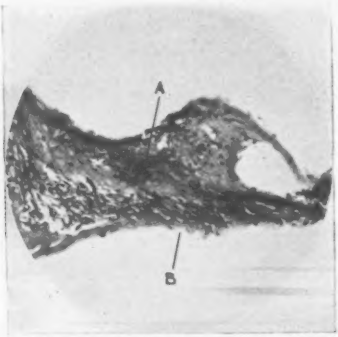



Fig. 11. Experiment 4A. Guinea pig No. 5. (A) Old small hemorrhage. (B) Hemorrhage undergoing organization.

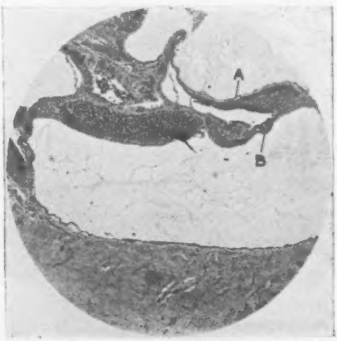



Fig. 12. Experiment 4B. Guinea pig No. 4. (A and B) Organizing hemorrhage.

placed in formalin 10 per cent and then Miller's solution. No apparent abnormality found in the sections.

*Experimental Control, No. 1A:* Stool extract of Mrs. E. N. Pig, Laboratory No. 216, weight 410 gm.

| | | |
|---|---|---|
| Feb. 9, 1928, 2 c.c. | Feb. 18, 1928, 1 c.c. | Feb. 26, 1928, 1 c.c. |
| Feb. 11, 1928, 2 c.c. | Feb. 19, 1928, 1 c.c. | Feb. 28, 1928, 1 c.c. |
| Feb. 12, 1928, 1 c.c. | Feb. 21, 1928, 1 c.c. | Mar. 1, 1928, 1 c.c. |
| Feb. 14, 1928, 1 c.c. | Feb. 23, 1928, 1 c.c. | Mar. 3, 1928, 1 c.c. |
| Feb. 16, 1928, 1 c.c. | Feb. 25, 1928, 1 c.c. | Mar. 4, 1928, 1 c.c. |

March 5, 1928, pig killed. Weight 365 gm. Autopsy normal. Ears and one eye placed in Miller's solution. Clump of red blood cells in cochlea in scala tympani.

*Experimental Control, No. 1B:* Stool extract of Mrs. E. N. Pig, Laboratory No. 248. Weight 410 gm.

| | | |
|---|---|---|
| Feb. 18, 1928, 1.0 c.c. | Mar. 9, 1928, 1.0 c.c. | Apr. 2, 1928, 1.0 c.c. |
| Feb. 19, 1928, 2.0 c.c. | Mar. 11, 1928, 2.0 c.c. | Apr. 4, 1928, 1.0 c.c. |
| Feb. 21, 1928, 3.0 c.c. | Mar. 13, 1928, 3.0 c.c. | Apr. 6, 1928, 1.0 c.c. |
| Feb. 23, 1928, 1.0 c.c. | Mar. 15, 1928, 1.5 c.c. | Apr. 8, 1928, 1.0 c.c. |
| Feb. 25, 1928, 2.0 c.c. | Mar. 17, 1928, 1.5 c.c. | Apr. 10, 1928, 1.0 c.c. |
| Feb. 26, 1928, 3.0 c.c. | Mar. 19, 1928, 2.0 c.c. | Apr. 12, 1928, 1.0 c.c. |
| Feb. 28, 1928, 1.0 c.c. | Mar. 21, 1928, 2.0 c.c. | Apr. 14, 1928, 1.0 c.c. |
| Mar. 1, 1928, 2.0 c.c. | Mar. 23, 1928, 2.0 c.c. | Apr. 16, 1928, 1.0 c.c. |
| Mar. 3, 1928, 3.0 c.c. | Mar. 25, 1928, 1.0 c.c. | Apr. 18, 1928, 1.0 c.c. |
| Mar. 4, 1928, 1.0 c.c. | Mar. 27, 1928, 1.0 c.c. | Apr. 20, 1928, 1.0 c.c. |
| Mar. 6, 1928, 2.0 c.c. | Mar. 29, 1928, 1.0 c.c. | |
| Mar. 8, 1928, 3.0 c.c. | Mar. 31, 1928, 1.0 c.c. | |

April 24, 1928, pig killed by chloroform and cutting throat. Weight 490 gm. Blood in tissues more profuse than by just cutting throat. Eyes and two ears in formalin and then Miller's solution. Sections: Fresh red blood cells in vestibule of the labyrinth.

*Experimental Control, No. 2A:* Stool extract of Mrs. A. F. Pig, Laboratory No. 218. Weight 280 gm.

| | | |
|---|---|---|
| Feb. 9, 1928, 2 c.c. | Feb. 18, 1928, 1 c.c. | Feb. 26, 1928, 1 c.c. |
| Feb. 11, 1928, 2 c.c. | Feb. 19, 1928, 1 c.c. | Feb. 28, 1928, 1 c.c. |
| Feb. 12, 1928, 1 c.c. | Feb. 21, 1928, 1 c.c. | Mar. 1, 1928, 1 c.c. |
| Feb. 14, 1928, 1 c.c. | Feb. 23, 1928, 1 c.c. | Mar. 3, 1928, 1 c.c. |
| Feb. 16, 1928, 1 c.c. | Feb. 25, 1928, 1 c.c. | Mar. 4, 1928, 1 c.c. |

On March 5, 1928, pig was killed. Weight 210 gm. Autopsy normal. Both ears and eyes in Miller's solution. Apparently normal histologically.

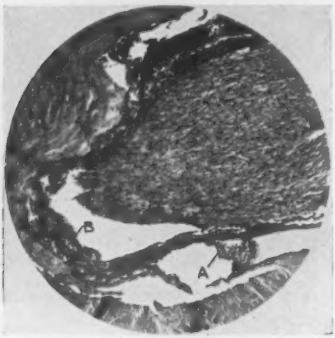



Fig. 13. Experiment 4B. Guinea pig No. 4. (A) Hemorrhage into dura surrounding auditory nerve. (B) Hemorrhage around the auditory nerve.


Fig. 14. Experiment 4B. Guinea pig No. 4. (A) Very old small hemorrhage into ligamentum spiralis. (B) Organized hemorrhage underneath organ of Corti.

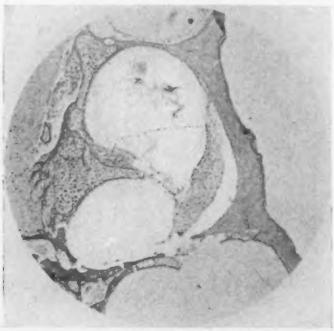


Fig. 15. Experimental control 3B. Guinea pig No. 249. No abnormality (75X).

*Experimental Control, No. 2B:* Stool extract of Mrs. A. F. Pig, Laboratory No. 217. Weight 320 gm.

Feb. 9, 1928, 1.0 c.c. Feb. 19, 1928, 1.0 c.c. Mar. 1, 1928, 1.0 c.c.
Feb. 11, 1928, 2.0 c.c. Feb. 21, 1928, 2.0 c.c. Mar. 3, 1928, 2.0 c.c.
Feb. 12, 1928, 1.0 c.c. Feb. 23, 1928, 3.0 c.c. Mar. 4, 1928, 3.0 c.c.
Feb. 14, 1928, 1.0 c.c. Feb. 25, 1928, 1.0 c.c. Mar. 6, 1928, 1.5 c.c.
Feb. 16, 1928, 2.0 c.c. Feb. 26, 1928, 2.0 c.c.
Feb. 18, 1928, 3.0 c.c. Feb. 28, 1928, 3.0 c.c.

On March 17, 1928, pig was killed. Weight 320 gm. after cutting throat. Eyes and ears placed in Miller's solution. Autopsy normal. Apparently negative histologically, except small clumps of fresh red blood cells in upper portion of cochlea in the scala vestibulae.

*Experimental Control No. 3A:* Stool extract of Mrs. A. S. Pig, Laboratory No. 221. Weight 305 gm.

Feb. 9, 1928, 2 c.c. Feb. 18, 1928, 1 c.c. Feb. 26, 1928, 1 c.c.
Feb. 11, 1928, 2 c.c. Feb. 19, 1928, 1 c.c. Feb. 28, 1928, 1 c.c.
Feb. 12, 1928, 1 c.c. Feb. 21, 1928, 1 c.c. Mar. 1, 1928, 1 c.c.
Feb. 14, 1928, 1 c.c. Feb. 23, 1928, 1 c.c. Mar. 3, 1928, 1 c.c.
Feb. 16, 1928, 1 c.c. Feb. 25, 1928, 1 c.c. Mar. 4, 1928, 1 c.c.

On March 5, 1928, pig was killed. Weight 285 gm. Autopsy normal. Negative histologically.

*Experimental Control, No. 3B:* Stool extract of Mrs. A. S. Pig, Laboratory No. 249. Weight 440 gm.

Feb. 18, 1928, 1.0 c.c. Mar. 9, 1928, 1.0 c.c. Apr. 2, 1928, 1.0 c.c.
Feb. 19, 1928, 2.0 c.c. Mar. 11, 1928, 2.0 c.c. Apr. 4, 1928, 1.0 c.c.
Feb. 21, 1928, 3.0 c.c. Mar. 13, 1928, 3.0 c.c. Apr. 6, 1928, 1.0 c.c.
Feb. 23, 1928, 1.0 c.c. Mar. 15, 1928, 1.5 c.c. Apr. 8, 1928, 1.0 c.c.
Feb. 25, 1928, 2.0 c.c. Mar. 17, 1928, 1.5 c.c. Apr. 10, 1928, 1.0 c.c.
Feb. 26, 1928, 3.0 c.c. Mar. 19, 1928, 2.0 c.c. Apr. 12, 1928, 1.0 c.c.
Feb. 28, 1928, 1.0 c.c. Mar. 21, 1928, 2.0 c.c. Apr. 14, 1928, 1.0 c.c.
Mar. 1, 1928, 2.0 c.c. Mar. 23, 1928, 2.0 c.c. Apr. 16, 1928, 1.0 c.c.
Mar. 3, 1928, 3.0 c.c. Mar. 25, 1928, 1.0 c.c. Apr. 18, 1928, 1.0 c.c.
Mar. 4, 1928, 1.0 c.c. Mar. 27, 1928, 1.0 c.c. Apr. 20, 1928, 1.0 c.c.
Mar. 6, 1928, 2.0 c.c. Mar. 29, 1928, 1.0 c.c.
Mar. 8, 1928, 3.0 c.c. Mar. 31, 1928, 1.0 c.c.

On April 24, 1928, pig was killed by anesthesia with chloroform and exsanguination by cutting throat. More free blood found in the tissues than by the other method. Eye and both ears in formalin and then Miller's solution. Weight on April 24, 1928, 440 gm. Sections: One temporal bone apparently normal (Figs. 15 and 16).

Other temporal bone shows osteomyelitis, with marked hypertrophic new bone formation in the middle ear, due to infection.

## CONCLUSIONS.

The stool of the patients whose hearing is becoming progressively worse is undoubtedly toxic to guinea pigs. It apparently produces hemorrhagic lesions in the cochlea, hemorrhages into nerves, around nerves and into the dura. These lesions were not found in the controls. The controls received more intense treatment than the animals that were injected with the extracts of stool from patients suffering with progressive deafness.

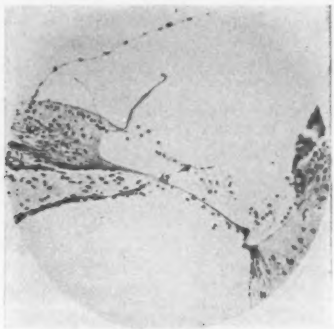


Fig. 16. Experimental control 3B. Guinea pig No. 249. No abnormality (230X).

## REFERENCES.

1. GOTTLIEB, M. J.: Constitutional Deafness. THE LARYNGOSCOPE, Vol. 38, p. 306, May, 1928.

2. KOPETZKY, S. J., and ALMOUR, R.: Blood Calcium in Cases of Undifferentiated Deafness. Collected Papers of the Oto-Laryngological Clinic of Beth Israel Hospital, New York, Bull. No. 3, 1927, p. 9.

3. KOPETZKY, S. J.: Further Studies Concerning the Nature of Progressive Deafness. Collected Papers of the Oto-Laryngological Clinic of Beth Israel Hospital, New York, Bull. No. 3, 1927, p. 23.

4. GOTTLIEB, M. J.: Contraction of the Visual Fields in Cases of Progressive Deafness. Archives of Oto-Laryngology, Vol. 8, pp. 12-25, July, 1928.

5. LEICHER: Zentrabl. f. Ohrenb., 27:4, 1927.

23 West 73rd Street.

## DEHISCENCE OF POSTERIOR WALL OF SPHENOID SINUS. REPORT OF CASE.*

DR. LOUIS K. ELFMAN, Philadelphia.

The paired sphenoidal sinus is located in what may be termed the danger position in the skull. Occupying a more or less central position dorsal to the posterior portion of the nasal cavity, it comes into intimate relationship, dorsally with the hypophysis cerebri and not infrequently with a portion of the brainstem; laterally with the dural cavernous sinus and its contained and related structures; cephalically with the optic commissure, and frequently with the hypophysis cerebri; caudally with the Vidian nerve, the choanae and nasopharynx; ventrolaterally with the optic nerve and ophthalmic artery.

The lateral wall of the sphenoid sinus is usually one of the thinnest walls of the cavity, often reduced by extensive pneumatization to a paper delicacy. Osseous dehiscences are occasionally found.

Zuckerkandl, Onodi, Spee, Meyer, Schaeffer and others have reported defects in the osseous lateral wall of the sphenoidal sinus. Dehiscences of the posterior wall have also been reported, but are not as common as those of the lateral wall.

I wish to report an interesting case of an asthmatic, who had a chronic ethmosphenoiditis, and who at operation showed a dehiscence of the posterior wall of the left sphenoid. As a result of this operation, the patient developed meningismus and encephalitis.

J. B., age 44 years, had been troubled with nasal discharge, headaches and asthmatic attacks for many years. The symptoms became worse during the past six years. He had been treated in various clinics in Philadelphia and New York, with no improvement. In 1928, he had a submucous resection done at the Bellevue Hospital, New York. Later, he was sent to a sanatorium for observation, as it was thought he might have tuberculosis. He was discharged as nontuberculous. He later drifted into clinics at the Jewish Hospital. An X-ray study of the chest in January, 1928, revealed fairly well marked fibrosis in both lungs, and several small bronchiectatic cavities. Repeated sputum examinations were negative for the tubercle bacillus. X-ray of sinuses showed some haziness in left ethmoids, sphenoid and antrum. Bronchoscopic examination showed an excess of thick, tenacious mucus in bronchi and trachea. The mucous mem-

*Read before the Philadelphia Laryngological Society, Oct. 10, 1928.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication Oct. 26, 1928.

brane was reddened and granular. Examination of nose showed hyperplastic changes in both middle turbinates, some mucopus in naso-pharynx, and on anterior portion of left middle turbinate. Lavage of both antra gave return of clear fluid. The anterior tips of the hyperplastic middle turbinates were removed under local anesthesia, also the anterior tip of the left inferior turbinate. The patient's asthmatic attacks continued just the same. As pus was still present in the nose in ethmosphenoidal area, on left side, it was thought best to admit patient to the house and open ethmoid and sphenoid sinuses on that side.

On March 8, 1928, under local anesthesia, patient in the upright position, the remains of the left middle turbinate were removed, the ethmoid cells broken down and curetted, and the sphenoid sinus exposed. There was a dehiscence of the posterior sphenoid wall, a pulsating dura was visible at this point. The bleeding that followed soon stopped. Hyperplastic changes were observed in both the posterior ethmoid and sphenoid cells. However, the only curetting done was in the ethmoids. As soon as the pulsating dura was observed, the operation was stopped, a small piece of plain packing was placed in the tip of the nose, and patient was returned to ward.

The patient's behavior on the following day was queer. He seemed to be dazed at various times, complained of headache. There was no muscular spasticity. Late that evening he developed rigidity of the neck, with bilateral Kernig. Breathing became labored. Pulse was 68 and temperature rose to 100.4°, by axilla. Patient showed desire to pick at bedclothes. The clinical picture was typical of meningitis.

Blood culture was returned negative after three days. Two specimens of spinal fluid in the week following operation showed no cloudiness, 50 W. B. C. the first time and 3 W. B. C. the second, globulin was increased and sugar was present. Culture of both specimens were returned negative. Two eyeground examinations showed no gross pathology, and diagnosis of mild optic neuritis was made by ophthalmologist.

The patient had a stormy week following the operation. The highest temperature at any time was 100.4°. By the end of the week temperature and pulse were normal. The patient still seemed to be a little flighty in speech and thoughts.

At the end of the second week, the patient was able to go about the ward unassisted. Temperature was still normal.

Fifteen days after the operation the patient had a relapse. Began picking at his clothing, was lethargic, unable to walk around unassisted. Complained of headaches again, and it appeared as though he had a mild encephalitis now. These symptoms cleared up gradu-

ally, and 22 days after the operation, the patient was discharged. At the time of discharge, his mind showed some evidence of confusion. He was taken to a private sanatorium by his nephew, for further convalescence.

We have lost sight of this patient the last few months, but our social service department reports that he is being treated in one of the chest clinics in the city; and also reports that his asthmatic attacks are not as severe as they have been in the past.

In conclusion, this case presents several outstanding features:

*1.* An anatomical abnormality—dehiscence of posterior wall of sphenoid, which is uncommon.

*2.* Extreme care should be exercised in surgical procedure on the sphenoid sinus, due to the relationship of the posterolateral wall with the cavernous sinus.

*3.* Due to irritation of the dura as a result of operative procedure and dehiscence of posterior sphenoidal wall, the patient developed a severe meningismus, and mild encephalitis, with a fortunate recovery.

*4.* Asthmatic symptoms are still present, although attacks are not as frequent, nor as severe.

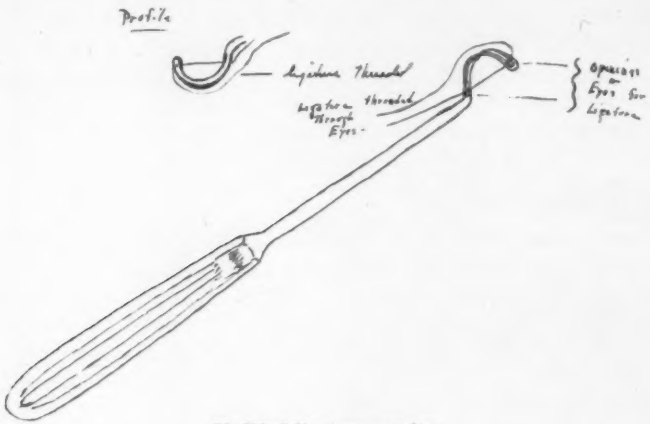
Before closing my paper, I would like to thank Dr. Herman B. Cohen for his co-operation in the handling of this case.

2008 Walnut Street.

## A MODIFIED LIGATURE CARRIER.*

DR. FREDERICK T. HILL, Waterville, Me.

Sometimes in doing a jugular ligation, using the Tobey incision, there is difficulty with the retraction in a person with a fat, thick neck. The ordinary ligature carrier, being of rather a wide curve, may not readily pass around the vein but the point may become engaged in the tissues so that the ligature may not be easily picked up. In order to overcome this difficulty the curve of the ligature carrier has been made sharper so that it will hug the vein closely



Modified ligature carrier.

and pass freely up through the small incision. To facilitate picking up the ligature after passage around the vein, a second eye has been put in the butt of the arm, through which one end of the ligature is threaded. When this is drawn taut it lifts the ligature away from the carrier like the string of a bow, so that it may be quickly picked up with the forceps. This instrument is made by Codman and Shurtleff, Boston.

Professional Building.

*Read before the American Laryngological, Rhinological and Otological Society, Washington, D. C., May 3, 1928.

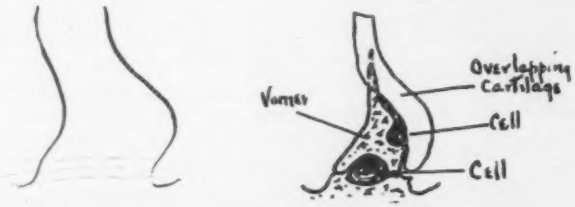
Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication Sept. 15, 1928.

## CELLS IN THE VOMER.

Dr. Douglas Macfarlan, Philadelphia.

The following case is cited as a curiosity, and with the hopes that by some chance it may find corroboration in the experience of other workers.

In the course of a septum operation for the removal of a deflected triangular cartilage and a bony spur, there was found two cells in the vomer. Both were situated about three-quarters back. The first was one-half within the crest of the vomer, and one-half within the cartilage above it; the second lay beneath it and entirely within




Cells in the vomer.

the vomer. Each had a mucous membrane lining. No orifice was found in the upper, but this was probably due to the damage of the cell during the operation. In the lower one, a minute opening was found running upward, backward and outward under the spur.

The cells had all the appearance of a familiar aeration cell, but why they should be found in this region is puzzling.

There was nothing to suggest a cystic degeneration, and there was no secretion within the cell.

I have not heard of any anomalies of this nature in this region.

1805 Chestnut Street.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication Oct. 27, 1928.

## BOOK REVIEW.

**Hay-Fever and Asthma,** Their Cause, Prevention and Treatment. By Ray M. Balyeat, M.A., M.D., F.A.C.P., Instructor in Medicine and Lecturer on Allergic Diseases in the University of Oklahoma Medical School; Consulting Physician to St. Anthony's Hospital and to the State University Hospital; Member of the American Association for the Study of Allergy; Vice-President, Oklahoma Academy of Science; Director, Balyeat Hay-Fever and Asthma Clinic, Oklahoma City, Okla. Illustrated with 76 engravings, including 2 in colors. Second edition, revised and enlarged. Philadelphia: F. A. Davis Company, 1928. Price $3.00 net.

The fact that this book is written in a simple, clear and understandable language so that the layman can easily follow its gist does not detract from its importance to medicine as most of the medical profession can benefit from such a clearly written account of a rather slightly known subject.

A very well taken point is (page 26), "The individual inherits the tendency and not the specific state. The transmission of ability to become specially sensitive seems to follow Mendel's law."

"Inheritance is a chief factor in determining whether or not a patient will develop hay-fever or asthma and it governs very largely the time in life when the symptoms will appear."

The author very definitely points out how mistaken we have been on insect-pollinated plants causing symptoms and, in his experience, goldenrod, sunflower, rose, field daisy and dandelion play a relatively small part in the cause of hay-fever.

A rather comprehensive geographical division of the United States is made, with full tables of pollens and their origin as well as their dates of bloom. A more detailed account of conditions about Oklahoma is given and reports of the author's experimental research are given.

The chapter on "The Relation of Domestic Animals and the Causes of Hay-Fever and Asthma" gives a concise account of which animals are likely to carry pollen. Fur, as worn in garments, has been shown to often be the cause by virtue of the dust in the hairs.

Cosmetics, which nearly all contain orris root, may give predisposition or actual symptoms of hay-fever and asthma.

The author makes mention of foods as a predisposing cause and claims that asthma in infants in 15 per cent of cases may be traced to some food.

Balyeat claims that asthma is not due to bacterial infection. He used to think that about 25 per cent of asthmatic cases were due to bacteria when he could find no sensitive cause, but he now feels that infection plays no role in the cause of asthma.

Methods and technique of dermal and intradermal tests are clearly enunciated and the results interpreted.

The treatment and care of these patients is gone into and an honest opinion of how much relief is to be expected is given by the author. M. F.

---

## IN MEMORIAM.

Dr. Eldred B. Cayce, of Nashville, Tennessee, died on Dec. 4, 1928, at the age of 52 years.

Dr. Cayce graduated from University of Nashville Medical Department in 1910. He enjoyed the respect of his fellow practitioners and his loss will not only be felt by his friends, but by the profession at large.

# International Digest of Current Otolaryngology

*Editor:*
Dr. Maxwell Fineberg, St. Louis.

*Collaborators:*
Priv. Doz. Dr. J. Berberich, Frankfurt a/M.
Mr. W. S. Daggett, London.
Priv. Doz. Dr. G. Kelemen, Budapest.
Dr. D. E. Staunton Wishart, Toronto.
St. Louis Jewish Hospital E.N.T. Journal Club.

The editorial staff of this column wishes to voice its appreciation for the comment expressed concerning this new Digest of Current Otolaryngology. Readers are again invited to submit for publication any news of scientific otolaryngologic interest. The purpose of this column is to stimulate interest in scientific research and to keep our readers informed in a brief way of what is being done along these lines in various centers of the world. We invite brief preliminary reports of research or new ideas in this field with the understanding that such reports shall not prejudice subsequent complete publication. Enlightening group or society discussions on recent research or new methods of therapy, etc., are also invited.

We note with interest that the December, 1928, issue of the *Annals of Otology, Rhinology and Laryngology* discusses at some length the American Board of Otolaryngology. The history and purposes of the American Board are clearly outlined by its secretary, Dr. W. P. Wherry. Dr. Ross Hall Skillern contributes the part that he takes in the examination of the candidate. He is particularly interested in the character, scientific ability and personality of the candidate. Dr. Harris P. Mosher discusses the methods used in giving the didactic examination and discloses some of the reasons for his questions. The clinical examination is conducted by Dr. R. C. Lynch and he advises what factors influence him in basing the standing of the candidate. The examination in pathology is given by Dr. Joseph C. Beck and he very clearly outlines what he expects a candidate to know in response to his questions. Case histories are examined by Dr. Ralph A. Fenton and many valuable hints are given as to what is considered a good case report.

A resumé of what the American Board of Otolaryngology is and what it has done, is doing and hopes to do, is a very timely contribution to the literature. It undoubtedly is the aim of the Board to

eventually have every specialist in this field a member of this Board and those of us who have only vague information concerning this group can derive much information from a careful perusal of this series of articles. Prospective candidates are advised to keep this issue of the *Annals of Otology, Rhinology and Laryngology* on file as it undoubtedly will be of great help to them in planning their studies for the examinations.

---

It is noted in the January issue of the *Journal of Laryngology and Otology* that Drs. Fraser and Logan Turner have relinquished the editorial duties to Drs. Howarth, Negus and Watkyn-Thomas.

With the inspiring principles of such antecedents and the capabilities of the present editorial staff we feel sure that the *Journal of Laryngology and Otology* will not only maintain its usual high standards but will ever strive to attain new heights.

---

In this issue of THE LARYNGSCOPE is found the full text of the Sir Felix Semon lecture delivered before the Royal Academy of Medicine, November 1st, 1928, by Prof. M. Hajek, of Vienna. It is indeed a fitting honor to Prof. Hajek to be called upon to deliver this lecture. Dr. Semon was one of the greatest British laryngologists and it is only proper that one of the greatest living laryngologists be called upon to do honor to his memory.

THE LARYNGOSCOPE takes great pleasure in republishing from the January, 1929, issue of the *Journal of Larynogolgy and Otology* the complete text of Prof. Hajek's address.

---

CHOLESTERIN, METABOLISM AND OTOLOGY—Fineberg, St. Louis; Berberich, Frankfurt a/Main.

The relationship of arteriosclerosis, hypertension, chronic nephritis and apoplexy to so-called senile deafness is shown to be closely bound to a hypercholesterinemia and equal bilateral, peripheral, internal ear deafness, is shown to clinically exist in these cases. Histologically, it was possible to show lipoid or fatty deposits in characteristic locations of the temporal bones in previously examined clinical material of this sort. Further evidence in favor of this is presented by the experimental production of a hypercholestrinemia in rabbits whose temporal bones then showed the identical histological lipoid changes. (To be published shortly both in English and German.)

---

We are in recent receipt of the opening number of a new journal in our specialty, *Otolarynogologia Slavica,* edited by Prof. O. Kutvirt, of Prague, and numerous Slavonic collaborators.

This new journal is published by Bursik A. Kohout, Prague, Czechoslovakia, and appears to be a most interesting little reference monthly. The articles in the first number show that the type of work being done in the Slavonic countries is fully on a par with the work done in Europe and America. The literature appended to the articles shows numerous English, French and German references of the highest type. Articles are accepted in these languages.

---

We are in receipt of the new Japanese journal, *Oto-Rhino-Laryngologia*, edited by Prof. Ino Kubo in Fukuoka, Japan; we make no comment as we are unable to understand the language.

---

The Kansas City, Eye, Ear, Nose and Throat Society announces that it will be honored in its March Clinic Day by the presence of Dr. Samuel Kopetzky, of New York, and Dr. Allan Greenwood, of Boston. There will be a morning operative clinic and an afternoon diagnostic clinic. Dr. Kopetzky will operate mastoid cases only, and Dr. Greenwood will operate only glaucoma and strabismus cases. The exact day in March is not yet announced. Information may be obtained from the secretary, Dr. A. E. Eubank, 636 Argyle building.

---

This department is endeavoring to establish better contacts for those who desire postgraduate training either in America or Europe. We shall attempt to promptly report any news of such interest.

It is learned with regret that Prof. Marc Paunz, of Budapest, intends to relinquish his surgical training of American otolaryngologists. Prof. Paunz has taught more than 100 Americans in the past 10 years and those of us who were fortunate enough to obtain surgical training and experience through him hope that he will reconsider his decision and continue to teach.

---

Dr. Franz Hasslinger, assistant at the Hajek Klinik, of Vienna, is shortly expected to tour the United States, giving lecture courses in several of the larger cities.

Dr. Hasslinger, it will be remembered, is the designer of the "Directoscope" and the "Hasslinger Endoscopic Headrest." He will bring with him on his present trip one of his recent inventions, "The Electrical Phantom Larynx." This contraption is designed to teach laryngeal pathology and is foolproof as the student in pointing out the various parts of the larynx must make an electrical contact with his applicator on the exact spot, when a bell is automatically rung.

We can heartily recommend Dr. Hasslinger's courses, and wish him a pleasant sojourn in our country.